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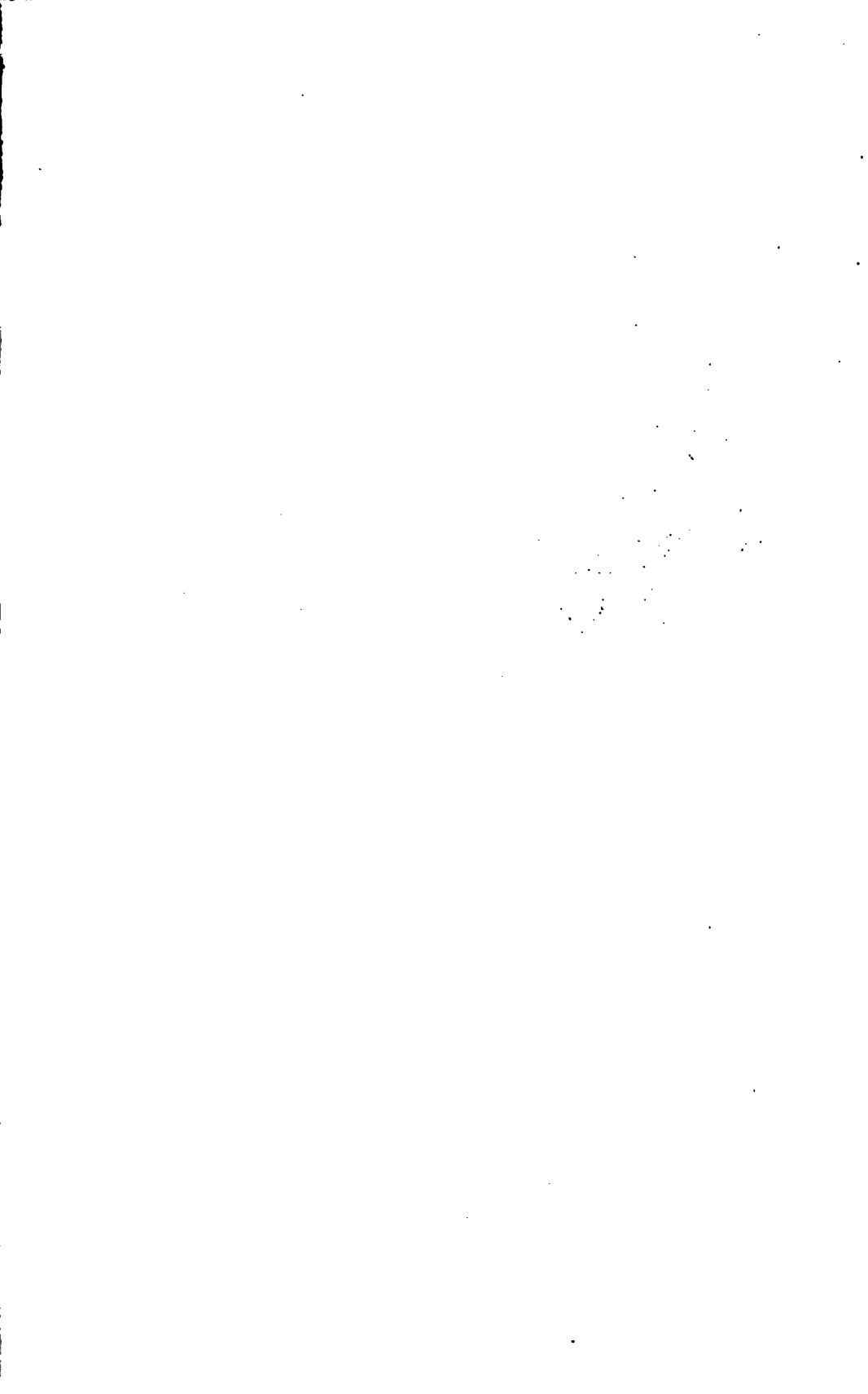
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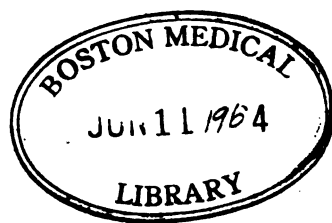
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THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ANNOUNCEMENT.

The members of the Editorial Staff, whose names will be found on the title page, have agreed to undertake the literary management of the **JOURNAL OF NERVOUS AND MENTAL DISEASE** for the year 1897.

It is their intention to make the **JOURNAL**, in all its departments, thoroughly representative of American Neurology, and to this end they ask for the active support of all interested in the study of nervous and mental disease. The editors have the promise of co-operation from so many well-known men in different parts of the country that they confidently expect that their efforts will be crowned with success. Special attention is to be paid not only to the publication of valuable original articles, but also to the Periscope Department and the Book Reviews. The **JOURNAL** is to be of use to the general practitioner as well as to the specialist.

This case was reported in a paper on "The complications of influenza." The doubt which existed in my mind as to the diagnosis, based on the fact that several weeks had elapsed between the disappearance of the influenza and the onset of the cerebral symptoms, would now be less than it was then, because, as I shall point out, the presence of this interval seems to be rather characteristic.

¹ *Deutsche Zeitschr. für Nervenheilkunde* 1895, vol. 6, p. 375.

² *Boston Medical and Surgical Journal*, October 6, 1892.

Since seeing this case I have met with another of still greater importance, which I shall report in the present paper. The practical interest attaching to my cases is that they confirm the important conclusion reached by Oppenheim; namely, that in spite of the extremely serious character of the symptoms, and although they indicate the simultaneous involvement of a number of important centres, complete or partial recovery is by no means uncommon.

The forms of encephalitis which are to be considered in this connection are those in which multiple foci of inflammation are met with, of variable extent, and characterized by the presence of signs of congestion, leucocyte infiltration, minute and even massive hæmorrhages, with greater or less destruction of nerve elements, but not by suppuration.* The same cause which produces the encephalitis, such as the influenza bacillus, may excite also meningitis, but when the meningitis becomes a prominent feature, as in the cerebro-spinal form, a new element of danger is introduced. It is also obvious that if a complete discussion of the subject from a pathological standpoint was to be attempted, other infectious diseases of the nervous system, such as disseminated myelitis and neuritis multiplex, would have to be considered.

The principal, though by no means the only, clinical symptom groups which deserve attention in connection with the subject of this paper are, (1) Wernicke's polio-encephalitis superior, in which the gray matter of the third ventricle, of the aqueduct of Sylvius, and of the fourth ventricle is chiefly involved; and (2) the form which was first distinctly pointed out by Strümpell[†] in 1890, where the lesions involve more especially the hemispheres, especially the cortex and the basal ganglia, or the cerebellum, and often both sides in a symmetrical fashion, causing a variety of symptoms, such as convulsions, aphasia, monoplegias and hemiplegias. Optic neuritis may be met with, and severe general symptoms, fever and coma, are always present. . Even the ocular paralyses may be

* It is to be borne in mind that, in view of the present inadequate state of our knowledge with regard to the anatomical changes present in the slighter cases of this class, and, on the other hand, in the light of our information with regard to the effects of bacterial and other toxins in general, we can by no means be sure but that some of the symptoms attributed to local inflammation are really due purely to toxine poisoning.

[†] In view of the early and important contributions of Leichtenstern to this subject Oppenheim designates these cases as of the Strümpell-Leichtenstern type. (See *Deutsche Med. W. Schr.* 1890 and 1892).

present, in connection with other symptoms, and thus the cases of this group touch hands with those of the other.

The Wernicke symptom-complex differs from that of the Strümpell form of encephalitis in that paralysis of the external muscles of the eye is present in a high degree, and that optic neuritis is of more common occurrence. The motion of the limbs may be impaired, and the speech affected, and general symptoms similar to those observed in the other form are likely to show themselves. The temperature may be elevated, or normal, or subnormal. The genesis of these diffuse cerebral symptoms has recently been studied anew by Jacobaeus,² who thinks that alcoholic neuritis with its cerebral and spinal complications plays an important part in their productions.

Most of the cases of this class certainly affect alcoholic subjects, and in them the outcome has almost always been fatal, though a few cases of other origin have ended in recovery. (See Jacobaeus, l. c.)

The clinical and experimental evidence is strong that these focal myelitic and encephalitic processes may be due to bacterial toxins of various sorts, and that the outbreak of encephalitis or myelitis may either attend or follow the primary manifestations of the infection, or may itself be the primary manifestation. A number of facts bearing on these points were cited in a summary of the results of infectious diseases, published by me in 1895³, and several new observations have appeared within the past year.

So far as human pathology is concerned, the poison of influenza appears to be the most active of the toxic causes of acute lesions, and the influenza bacillus has been found in the diseased foci, but the case to be reported to-day is especially interesting, because it seems to show that the mumps may be responsible for equally serious results. It should not, of course, be forgotten in this connection, that the real agent may be a secondary infection. Attention has indeed been called to the especial need of caution in this respect in the case of affections following the mumps, because there are some grounds for suspecting that the organism which causes this disease may not be, strictly speaking, bacterial, but rather protozoal.

¹ Ueber einen Fall von Polioencephalitis hæmorrh. sup. *Deutsche Zeitschr. f. Nervenheilkunde*, 1895, p. 334. (See also Kaiser, cited below).

² Relation of Infectious Processes to Diseases of the Nervous System. *Am. Journal of Med. Sciences*, 1895, Vol. 109, p. 254.

The cases of the form of encephalitis hitherto described by Strümpell, Leichtenstern, Fürbringer, and others, are, as Oppenheim justly observes, not well calculated to give a correct idea of the prognosis of the disease in question, because they were selected mainly in the interests of the pathological anatomy. A number of recoveries are, however, to be found among them.

Oppenheim himself adds six new cases, which in brief outline are as follows :

The first case was that of a girl of sixteen; the whole duration four months; the outcome favorable. The main symptoms were headache; motor and sensory aphasia, ushered in by speechlessness; optic neuritis. There was but trifling elevation of temperature.

The second case was that of a girl of only ten years; the duration of the illness about two months; the outcome favorable. The onset occurred three months after a slight attack of influenza, which may or may not have been responsible for the result. The symptoms were vertigo and prostration, passing rapidly into unconsciousness lasting three days; fever of short duration; loss of speech; stiffness of neck muscles; slight optic neuritis.

The third case was that of a woman of twenty eight. The duration of the illness, including a prolonged convalescence, was eight months. The onset was moderately rapid, and the first symptoms showed themselves in less than a month after an attack of influenza. The symptoms were, mental confusion; loss of speech; paresis of right arm and side of face, preceded by an epileptic seizure.

The fourth case was, again, that of a young girl of twelve. The duration of the illness was five or six months; the outcome unfavorable, and the cause unknown. The symptoms were paresis of the limbs and face, with partial degenerative reaction of the left facial muscles, ataxia, impairment of sense of position of the limbs, exaggerated knee jerk and ankle clonus, impairment of hearing, paresis of the palate.

The fifth case was that of a man of twenty. The duration of the illness was four months, but the recovery incomplete. Here also there were widespread sensory and motor symptoms, disturbed articulation, hemianopsia, double optic neuritis, tinnitus aurium.

The sixth case, that of a man of twenty-seven, is of especial interest, since it resembles that of my patient in the respects that complete ophthalmoplegia and difficulty

in deglutition were present, associated with motor disorder of the face and limbs, and impaired deglutition.

Since the publication of Oppenheim's paper some further contributions of importance have been made to both the clinical and pathological history of this affection. They reinforce the conclusion that the susceptibility of children is greater than that of adults, and also strengthen the view that the cause of the disease, toxic though it probably is, does not necessarily stand in a direct relation to an obvious infectious malady.

Freyhan's case is as follows: The patient was a boy of fifteen, and of good health, except for a tuberculous inflammation of the middle ear of long standing, which could not be considered as the cause of the subsequent illness, especially as the cerebro spinal fluid obtained by lumbar puncture was free from tubercular organisms. After a moderate attack of influenza the patient showed symptoms of cerebral disorder (transitory hemiplegia and apathy); and to these succeeded paresis of the right side of the face (of the peripheral type) and of the associated eye muscles; and, later, ataxia, vertigo, vomiting, and other symptoms. There was no fever at any time throughout the illness, and complete recovery occurred in the course of four months.

Nauwerck¹ reported the following cases with post mortem examination: The first is that of a girl fourteen years old, who was seized, during the influenza epidemic, but without having herself shown clear signs of infection, with fever which remitted and again increased, left-sided convulsions, vomiting, left hemiplegia. Consciousness became clouded quite early. The eyes were not affected. After a fortnight from the beginning of the illness, toward the end of which symptoms had occurred, restlessness and convulsions set in, with loss of consciousness. Death occurred the next day. At the post-mortem examination the cerebral substance was found soft, and numerous foci of hæmorrhagic softening were discovered in various parts of both hemispheres. No bacteria could be found by the usual methods.

The second is that of a girl of nineteen, who had been in excellent health except for a trifling cold in the head and pains in the limbs. The cerebral attack began suddenly, with headache, uncertainty of gait, faintness, vom-

¹ *Deutsche Med. W.schr.*, 1895, Vol. 7, p. 643.

² *Deutsche Med. W.schr.*, 1895, Vol. 7, p. 393.

iting, loss of pupillary light-reflex and slow pulse. That same night the pulse became still slower, fifty beats to the minute. The patient became dull and restless and died on the following day. At the post-mortem examination a hæmorrhagic focus of the size of a walnut was found in the right lobe of the cerebellum. This was surrounded by a zone of inflammatory softening; and bacteria, believed to be identical with the influenza bacillus, were found here, and also in the ventricular fluid, which showed signs of being of inflammatory origin. Nauwerck believes, with Leichtenstern, that these hæmorrhagic foci may sometimes be of embolic (bacterial) origin; and, with Pfuhl and many others, that the symptoms on the part of the nervous system may be the first sign of the infection.

Brie (Duren)* likewise reports a fatal case of unknown origin, with autopsy, the patient being, this time, a woman of thirty-six. The duration of the illness was nine days. The symptoms were loss of appetite, abdominal pains, trifling fever, headaches with increasing hebetude; later, rigidity of the neck muscles, dilatation of one pupil, coma, rapid increase of temperature up to the time of death. The post mortem examination showed the spinal cord, medulla oblongata, pons and cerebellum to be free from obvious lesions, and this observation increases the probability that the rigidity of the neck muscles observed in such cases is not especially a sign of meningitis. The cerebral hemispheres showed signs of general pressure—dryness of the surface, and flattening of the convolution—and within their substance, on both sides, numerous foci of hæmorrhagic softening small and large, were found.

Finally, a brief reference to two other important papers, bearing indirectly upon this subject, may be in place. The first is that of Kaiser," who discusses at length a fatal case in which the bulbar lesions of Wernicke's disease were associated with similar changes involving the nuclei of the spinal cord. The case is especially noteworthy for the present discussion on the following grounds:

1. The patient was a young and healthy man, not alcoholic and not sick with any infectious disease so far as known.

* *Allg. Zeitschr. f. Psych.* Bd. 53, p. 604.

¹⁰ Zur Kenntniss der Polienccephalomyelitis acuta. *Deutsche Zeitschr. für Nervenheilkunde*, 1895, Vol. 7, p. 359.

In this respect the case deserves to be ranked with those of the typical poliomyelitis of children and adults, and although the belief is growing that this latter disease is really of infectious origin, a belief strengthened by the occurrence of epidemics such as that recently described by Caverly,¹¹ in which cases of somewhat different character are apt to occur, side by side, yet it must be remembered that we have as yet no idea what the infectious agent is. In view of our ignorance on this point we cannot properly deny that on this unknown agent may be the effective causes, perhaps as a secondary infection, in a portion of the cases which we are inclined to attribute to some prevalent disease of a recognized sort.

2. The author discusses at some length the symptom of drowsiness, which may be present in a striking form before in the early stages of the illness, and shows that it presents itself not infrequently in these cases where eye paralysis is prominent, and seems to be associated with an involvement of the walls of the third ventricle. In the case observed by Dr. Drake and myself this somnolence seems to have been well marked for many days.

The second paper to which I would refer is that by Dinkler,¹² in which a case is described where vascular lesions, analogous to those described, occurred, again in a young child, without obvious cause except a severe blow on the head two and a half years before. In view of the length of this interval, no importance would have been attributed to this influence but that the child had been in an abnormal state ever since the injury. In view of this case, taken in conjunction with so many others where injuries have counted as partial causes of complex disorders of later occurrence, the possibility cannot be excluded that the mysterious susceptibility of the highly vascular nuclear gray matter was heightened by the fall on the head in this case, the more so that a general congestion of the cerebral cortex was found at the post-mortem examination.

The possibility that some toxic agent was also at work cannot be set aside, but none seems to have been apparent.

The case observed by myself is as follows :

¹¹ *Jr. of Amer. Med. Ass.* January 4. 1896.

¹² Mittheilung eines letal verlaufenden Falles von traumatischer Gehirnkrankung mit dem anatomischen Befunde einer Poliencephalitis hæmorrh. acuta. *Deutsche Z.schr. f. Nervenheilkunde*, 1895, Vol. 7, p. 465.

The patient was a healthy boy, nearly thirteen years of age, whom I saw in consultation with Dr. E. T. Drake, of Franklin, N. H., who has kindly given me his notes of the case to supplement my own.

In November, 1895, he passed through a moderate attack of mumps, and had apparently been entirely well for ten days before the present illness began. On December 28, 1895, he woke up feeling poorly, and went into his brother's room saying that he thought he had "a squint in one eye," in spite of which, however, he went out of doors and followed his usual occupations. The next morning he had a partial ptosis of both eyelids, especially the right, and impairment of motion of the right eye, which is said to have turned inward. He also became slightly deaf at this time, though at the moment not much was thought of it. He also complained of pain in the forehead, which developed into an intense and persistent headache, and of dullness and drowsiness which soon became so marked that he could be aroused only with difficulty, though it could be done. Two days later he was seen by Dr. Drake, who found his temperature to be 100° F. By this time the paralysis of the eyes, and the deafness had increased, and for the next week all his symptoms grew steadily worse. At times he would be slightly delirious, but generally his consciousness was fairly good after he had been fairly aroused. Sensations of numbness were complained of, referred to the left arm and leg, and these limbs were both weaker than those of the right side, though there was no paralysis. In fact, he had to be restrained by force from getting out of bed and could stand erect on both feet. The temperature ranged from 99° to 100°; the pulse from 70 to 90.

I saw him on January 1, 1896. He was then lying in bed, picking restlessly at the bed clothes like a typhoid patient, but using the right hand more than the left. His nose was filled up with mucus which, apparently, he had not strength enough to expel. His deafness seemed almost complete. Both eyes were motionless and fixed in the median line. The pupils were rather small and did not respond to light. There was ptosis of both lids. The power of swallowing was considerably impaired. Thus, milk would run out from his lips, and the reason seemed to be because the latter could not be pressed firmly together. It was, indeed, noticeable that there was paresis of the lower facial muscles, especially on the left side.

An ophthalmoscopic examination showed double optic neuritis of moderate intensity. He could not be made to respond to questions; resisted attempts to instil cocaine into the eye, and gave indications of pain when pricked. The face was somewhat flushed and rather spotty; the ears rather red. The knee jerks and wrist jerks were absent on both sides. The plantar reflexes were present, but the right was greater than the left. The abdominal reflexes were present. So far as could be judged by the motions of the patient, the sensibility of the skin over the whole left leg, the left side of the body, and the left arm was noticeably less than that of the corresponding parts of the right side, but no difference could be made out with certainty between the two sides of the face.

The treatment followed was mainly expectant, care being taken to get in a good quantity of nourishment and full doses of strychnia. The case at that time looked very serious, but with the remembrance of Oppenheim's reports in my mind, I ventured to hold out the possibility of a favorable outcome, at least as regards life. Since then I have learned through three letters and one interview with Dr. Drake that this hope has been more than justified. On January 4th, he was reported as free from fever, but with total loss of hearing and almost complete loss of eyesight, and the pupils were more contracted than when I saw him and did not respond to light. The right eye could be moved to a slight degree. On January 8th, he showed more consciousness and recognized Dr. Drake at his visit. He could raise the lids a little and apparently heard somewhat better. January 30th, Dr. Drake wrote, "Our boy is doing finely. His hearing is nearly, if not quite normal. He seems to have perfect control of the left eye and lid, and the sight is nearly normal. He can open the right eye only about two-thirds, and the ball turns out, and the sight is not yet normal. When I ask him to fix his eyes upon my finger at his left, lateral nystagmus is noticeable (in right eye only). When he uses both eyes he sees double and the right object is the more distinct. The appetite is good and he sleeps well. His speech is quite natural and mind clear. Memory is good. Fainted about a week since while sitting up in bed drinking milk. Sits up half an hour a day. The boy is anxious to get about the house."

On March 18th, that is, about three months after the onset of the illness, the boy was brought to see me with his mother on their way to the South, and I obtained the following additional facts:

His eyesight was still somewhat defective, though improving. Double vision was still present and tests showed that the vision of the right eye was slightly deficient and that of the left normal. The motion of the right eye was also still imperfect, though not wholly lost for either direction. That for the left eye was good. The pupils on both sides failed to respond to light except very slightly. The left was of moderate size, slightly irregular in outline; the right somewhat smaller than the left. Traces of optic neuritis were still noticeable on both sides. The hearing was thought by the patient to be as good as ever, but tests with the watch showed that his hearing distance was but five inches on the left side as compared with five feet on the right. The bone conduction was about equal for the two sides. The grasp of the right hand was somewhat weaker than that of the left. The knee jerk was lacking for the right side and normal for the left. He looked strong and well and his mental condition was excellent. His weight was fifteen pounds greater than before his illness. In spite of this, on the whole favorable, condition, he had shown one symptom, for the outcome of which I shall watch with interest. Ever since his illness, namely, he had had epileptiform attacks, which at first recurred many times a day, sometimes he would even have several attacks in the course of a few minutes. They last from five to fifteen seconds, and during that period there is twitching of the right arm and the right side of the face. He is unable to speak, or talks gibberish, and is evidently dazed though he does not wholly lose consciousness. Of late these attacks had been less frequent.

A recent letter from Dr. Drake, of the date of November 16th, 1896, describes the present condition as follows: "The diplopia is still present, but he is learning to ignore the left and fainter image, so that he can read and study without much annoyance. As regards his hearing, he can hear the tick of a watch with the right ear at thirty inches, with the left at twenty inches. He is entirely free from the epileptiform seizures, but my impression is that he is not so strong mentally as before. A sister says, 'he does many queer things; is destructive; is not irritable nor quick to take offence, but does such funny things.'"

The remarkable number, character and variety of these symptoms, involving epileptiform seizures, hemiparesis, and coma, ophthalmoplegia, temporary loss of

hearing, and impairment of deglutition and loss of knee jerks surely points to wide-spread lesions, reaching from the cortex to the medulla, and the general resemblance of the symptom complex to that present in the cases described and collected by Oppenheim justified, at least provisionally, the diagnosis of disseminated encephalitis. The extreme loss of hearing seems to be an exceptional but not a wholly new feature. Some degree of deafness was present in one of the cases cited, tinnitus in another, and word-deafness in a third. From the early occurrence of the symptom and its association with the ophthalmoplegia, I am inclined to refer it to an affection of the auditory nerves or centres, rare as this lesion, for some reason, seems to be. The relation of this case to the mumps must be left in question.

It is evident that the symptoms in different cases of this affection must differ a great deal in view of the variability of the seat of the lesion and the nature of the influence which causes it. There are, however, certain clinical facts which it is important to bear in mind: (1) For some reason, a large proportion of the patients (omitting the alcoholic cases) have been children. (2) The temperature, although usually high, is occasionally but little elevated and may be even below the normal. This is said to occur particularly often with the alcoholic poli-encephalitis of Wernicke, but the same fact has been observed with cases of other sorts, so that the alcohol is probably not itself the cause of the lowered temperature. (3) Even when the encephalitis follows an acute disease, a period of one to several weeks may pass before the outbreak. This leads naturally to a closer consideration of what the real cause of the disease may be. The principle antecedents, beside the influenza, have been alcohol, sulphurous acid, fright, trauma, puerperal disease, cerebro spinal meningitis, ulcerative endocarditis, but sometimes no morbid antecedents could be discovered. Oppenheim raises the question whether a light attack of influenza which had been recovered from some weeks before could have had to do with the onset of the affection in one case, and thought that this was not impossible. The case which I reported in 1892 was that of a man in early middle life, who had had in January a well-marked attack of influenza, during which intense headache was a prominent symptom. From this he apparently recovered entirely, so that he had returned to work, though still troubled more or less with headache, when, some eight

weeks after the occurrence of the primary seizure, the cerebral symptoms came on. The actual mode of onset was quite interesting. The patient was seized with a violent fit of sneezing while at his work, and before this was over, he found that he could neither hear nor see. The hearing quickly returned, and the vision almost entirely, but not wholly. From that time on he was confined to the bed with intense headache and delirium. Two weeks later he had an attack of left hemiplegia and slight impairment of speech. The hemiplegia began to lessen after two days and at the end of a week had practically disappeared. At the time that I saw him, which was five weeks after the beginning of his illness, although he was slowly mending, I found double optic neuritis with swelling and hæmorrhages. In the end, however, he recovered.

Fürbringer speaks of a case of encephalitis following scarlet fever where the cerebral symptoms showed themselves in the fifth week after the beginning of the primary illness.

In all these cases it is, of course, quite possible that we may have to deal with a secondary infection, or with some unappreciated cause, but it is also true that, on the one hand, the bacterial toxines may linger in the tissues far longer than one would conceive to be probable, and, on the other hand, that the outbreak which eventually comes may have been for some time closer to the surface than we had supposed. A large number of facts could, for example, be cited to show that in many cases of toxic neuritis due to the chronic influence of some poison, the final outbreak had long been prepared for by specific nutritive changes, some of which would probably have been recognizable under the microscope, just as in Gombault's lead poisoned guinea pigs signs of degenerative neuritis were discovered, though no paralysis had been present.

In this connection I wish to refer to a case (not hitherto reported) of acute ataxia of all four extremities, of sudden onset and ending in partial recovery, the patient having been a healthy man in middle life, an intelligent mechanic. The case was evidently one of the kind which has been described by Leyden as *acute ataxia*, and I mention it partly because these cases are probably due to an acute disseminated myelitis, analogous to the disseminated encephalitis, but sometimes terminating in sclerosis; partly in order to call attention to the fact that this

patient was taken ill during a severe epidemic of influenza, though without having shown any of the catarrhal symptoms. Of course it is impossible to assert with confidence that the influenza poison was responsible for his final illness, but there are indications that just as diphtheritic paralysis may occur where little or no recognizable sore throat has gone before, so, also, something similar may be true for the case of influenza. (4) Must we assume that in cases presenting the clinical aspect of toxic encephalitis all the symptoms are referable to recognizable lesions, even such trifling ones as those recorded in some of the post-mortem reports above referred to?

It seems to me that this question is distinctly to be answered in the negative. Chemical changes in nerve-cells, of toxic origin—using that term in its widest sense—are fully capable of leading to abnormal activity or to paralysis, and even when gross lesions are found, their importance often consists as much in affording a sign that the conditions necessary for toxic influence were present, as in defining the actual cause of the symptoms. Disordered vaso motor action is another partial cause.

It seems to be certain that some of the irritative symptoms usually considered indicative of inflammation of the cerebral membranes, in this class of cases, such as spasm of the muscles of the neck and back, may occur without this lesion;¹ and even such paralyzes as follow diphtheria may be independent of any gross change that we can as yet discover. I would call attention in this connection to two cases of personal observation. The first is that of an elderly lady recorded in the paper already referred to,² who, during a convalescence from influenza, suffered from a series of frequently recurring typical Jacksonian convulsions, at first unattended with loss of consciousness, but ending on the third day in hemiplegia and death, where the autopsy and microscopic examination revealed nothing but the signs (as I finally concluded) of universal oedema cerebri. The second is a case of a kind to which Dr. Wm. Osler referred in a discussion as to the nature of "typhoid spine" in the *American Journal of Medical Sciences* for 1894, (vol. CVII., p. 23). The patient was a middle aged man,

¹ In the discussion of this paper Dr. Osler especially called attention to this fact.

² On the Nervous Complications of Influenza. *Boston Medical and Surgical Journal*, October 6th, 1892.

seen in consultation with Dr. Hodgdon, of Arlington, who, during a protracted convalescence from a severe attack of typhoid, suffered extremely from violent and painful spasm of the extensor muscles of the back, brought on by the slightest movement, and suggesting the diagnosis of meningitis, which was, however, quickly abandoned. Eventually, while sitting up to take some food, he was seized with an attack of vomiting and instantly died. An autopsy, made by Dr. W. F. Whitney, showed a high degree of anæmia throughout the spinal cord, medulla oblongata and membranes, but no trace of gross lesion.

A CLINICAL STUDY OF INFANTILE HEMIPLEGIA.

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DR. CHARLES K. MILLS, during his term of service at the Pennsylvania Training School for Feeble-Minded Children, has permitted us to examine with him and to publish the following cases of infantile hemiplegia. We can not adequately express our appreciation of this and other acts of kindness on his part, and beg that he will accept this feeble acknowledgment.¹

Few subjects have been more carefully studied than that of hemiplegia, and in the study of the infantile forms of paralysis American authors have distinguished themselves almost more than in any other branch of neurology.

It is, perhaps, well to state at once that in devoting our attention to infantile hemiplegia we may be exposed to a criticism similar to that bestowed by Sachs¹ upon the work of Freud and Rie. Infantile hemiplegia should not be separated from infantile diplegia or paraplegia; they are all three merely clinical manifestations of similar lesions of which the location alone is different, and a lesion which, in a given case, has caused hemiplegia might have caused diplegia had it been more extensive. Merely for the sake of convenience we have selected a certain group of cases and have

¹ Sachs, Volkmann's Sammlung klin. Vorträge, 1-29, p. 438.

² The excellent photographs (numbers 2, 3, 4 and 5) have been taken under the supervision of Dr. Frank White. For photograph number 6 we are indebted to Dr. Robert Formad, and for number 7 to Mr. G. C. Bird, Jr. To Drs. Barr, Llewellyn, White and Morris we desire to express our appreciation of most valuable assistance.

attempted to study the clinical pictures presented. The time at our disposal was limited, and it was necessary to make some classification in order to use the rich material afforded by the Pennsylvania Training School for Feeble Minded Children.

We have examined in detail thirty-three cases of hemiplegia from the Elwyn institution; twenty-three of these were girls, ten were boys, seventeen were cases of right, sixteen of left hemiplegia. A more equal division in regard to the side affected could not be desired.

In six cases facial paralysis in the distribution of the lower branch of the seventh nerve was observed; in three the naso-labial fold on one side was less prominent. König¹ has confirmed the statements of Freud and Rie² in regard to the frequency of involvement of the muscles in infantile cerebral paralysis innervated by the seventh nerve. These authors have noticed some involvement of the facial muscles in twenty-five of their thirty-five cases.

We have not found the muscles supplied by the motor branch of the fifth affected. These are sometimes paralyzed in hemiplegia, as personal examination of cases in the adult has taught us.

In four of our patients the tongue was found paretic on the side of the hemiplegia, and in one there was marked unilateral atrophy.

The diminution in the size of the thorax is very common, but lessened expansion on the paralyzed side was not observed. In sixteen out of the twenty-eight cases which could be examined the thorax on the paralyzed side was smaller.

In some patients the difference was as great as an inch and a quarter, or even an inch and three-quarters. Great care was observed in order to avoid mistake, and where the difference was very little it was not recorded. The thorax does not appear to be smaller when the upper extremity is well formed, but in a few cases we have found the upper extremity much affected, and yet the thorax was of normal development.

Impairment of the intellectual faculties, to a marked degree, was noticed in thirteen cases. In some of the other patients the intellect was not of a high grade.

¹ König, *Neurologisches Centralblatt*, 1895, p. 797.

² Freud and Rie, *Klinische Studie über die halbseitige Cerebrallähmung der Kinder*.

Sachs⁴ states, in his recent work, that he has found idiocy in thirteen per cent. of the subjects of hemiplegia. This seems to us, judging from our cases, which may, perhaps, offer an unfair standard, rather an under-estimate. The degree of idiocy does not seem to bear any constant relation to the degree of the hemiplegia.

In none of these cases were the muscles of the neck involved, nor are we aware that this involvement has ever been observed. The only explanation is that the innervation of the muscles of each side of the neck from both hemispheres of the brain is so perfect that an unilateral cerebral lesion can cause no paralysis of these muscles. The same is true of the muscles of the larynx. This is an opinion held by Krause, Semon and Horsley, and Broeckart and recently supported by Simerka.⁵

In progressive muscular atrophy the upper part of the trapezius muscle commonly survives after the lower portion of it has been destroyed. There may be an explanation for this as given above. The ganglion cells in the anterior horns of the spinal cord which supply this part may have a more perfect double innervation, and through this stimulus preserve their vitality longer than do those which supply the lower part of the muscle. Marinesco and Goldscheider have advanced the theory that motor cells preserve their vitality only so long as they receive normal impulses.



FIG I.

The muscles of the two sides of the neck move in more perfect unison than do those of the back and shoulder girdle, and the upper part of the trapezius is constantly employed in maintaining the erect position of the head.

We have not found aphasia, even in right hemiplegia, in any case in which the impairment of the intellect was not of high degree, except in one case where the patient was both deaf and dumb. It is well known that aphasia, either sensory or motor, occurring in childhood, usually completely disappears. Wernicke's case, quoted in the

⁴ Sachs, *Nervous Diseases of Children*, p. 538.

⁵ Simerka, *Revue Neurologique*, June 15, 1896.

recent excellent thesis of Mirallié⁸, tends to confirm the view that the speech centres of the right hemisphere become active when the normal centres are destroyed. He reported the case of an adult who was aphasic and paralyzed on the right side. Speech was regained. A second attack of apoplexy reproduced the aphasia, and this time it was associated with left hemiplegia. At the autopsy an old lesion occupied the centre of Broca in the left hemisphere, and a more recent one was found in the foot of the right third frontal convolution. Oppenheim⁹ holds the opinion that the speech centres of the right side of the brain, even in right-handed persons, are partially developed. A most interesting statement is made by Sachs.¹⁰ According to him, the proportion of aphasia in children with left hemiplegia is relatively large, and the explanation which he offers is that at this period of life the differentiation in the hemispheres is not as perfect as in adult life.

Shortening and general diminution in size of the paralyzed limbs was found in thirty-one of the cases, and, as has been recorded by other writers, this was always greater at the peripheral extremities. We will not attempt to give figures in regard to the length of the limbs. Féré¹¹ has recently studied this subject in a number of cases.

For the determination of the length of a limb Dr. Llewellyn suggests the use of the instrument employed by shoemakers in measuring the length of the human foot. It may be employed with advantage (see Fig. 1).

In eight of these cases of right hemiplegia, where the intelligence was of sufficient grade to permit a test of the writing to be made, it was found that spontaneous writing was possible in five cases; one patient could print a few words, another could copy script as script, another could not write at all. Imperfect education may have been the cause in these three cases.

Sensation was not altered in any of our cases, nor was it in any of those reported by Osler.¹² Oulmont has, however, observed disturbance of sensation, and Raymond has seen hemianæsthesia once (quoted by Marie).

Epileptiform convulsions have been observed in

⁸ Mirallié, *De l'Aphasie Sensorielle*.

⁹ Oppenheim, *Die Geschwülste des Gehirns*, p. 85.

¹⁰ Sachs, *Nervous Diseases of Children*, p. 532.

¹¹ Féré, *Revue de Médecine*, No. 2, 1896.

¹² Osler, *The Cerebral Palsies of Children*.

twenty-two cases. In five more convulsions occurred in infancy. Sachs and Peterson believe that forty-four per cent. of all cases of infantile paralysis develop epilepsy, and Sachs thinks that a very fair proportion of cases of ordinary epilepsy are developed in connection with infantile palsies, which may not be recognized. The percentage in our cases is much greater than forty-four. In fully ninety-five per cent. the initial lesion of infantile palsy is in or upon the cortex, according to Sachs,¹¹ and is usually hemorrhage, thrombosis, or embolism. This causes a development of secondary sclerosis throughout the cortex with rather surprising rapidity. The frequency of the cortical location explains the frequency of the convulsions. According to Marie¹² the epilepsy of infantile hemiplegia is peculiar; there is an absence of foam and blood from the mouth, as the tongue is rarely bitten, very seldom are there involuntary evacuations, the attacks end abruptly, there is no period of stertor or coma, and when convulsive movements have ceased consciousness returns, and the child resumes its play as if nothing had happened. After the epilepsy has lasted for a long time these differences may not be noticed.

Gowers¹³ states that ultimately the fits may be preceded by an aura identical with some of the common warnings of idiopathic epilepsy. We have found that in at least six of our patients the approaching attack could always be foretold by an aura. One patient has paræsthesia of the throat, and pain in the region of the heart; another has spasmodic movements of the body; another perspires freely on the head and shoulders, and has a rush of blood to the head; three have peculiar sensations located in the stomach. Some of the other patients are of too feeble intellect to make any statement in this respect.

The etiology of these cases is very difficult to obtain. Carefully worded blanks are sent to the relatives of the children admitted to the institution, but they are often imperfectly filled out. In many cases convulsions occurring in the first few months after birth are given as the cause. It seems to us these should in general be

¹¹Sachs, *The New York Medical Journal*, Feb. 1892.

¹²Marie, *Dictionnaire Encyclopédique des Sciences Médicales*, 1888.

¹³Gowers, *Diseases of the Nervous System*, Vol. II., p. 459 (English edition).

¹⁴Freud, *Zur Kenntniss der cerebralen Diplegien des Kindesalters*, p. 60.

regarded as the first manifestations of a previous lesion, and in this we incline toward the opinion of Freud¹⁶, Dercum,¹⁷ etc. Dercum believes also that permanent changes in the nutrition of the cortex may occur in some cases from prolonged spasms. Osler and Sachs are much in favor of the view that convulsions may be the cause of hemiplegia, and their arguments are forcible. In one of our cases we find scarlet fever given as a cause, in another "a congestive chill, followed by apoplexy and unconsciousness for four days;" in another "brain fever," in another "catarrhal fever," in another small-pox. Lovett could in one case only associate the beginning of the paralysis with an acute infectious disease, and that was diphtheria. Imogene Bassette¹⁸ has published a number of cases of paralysis following nearly all the infectious diseases of childhood, although in many of her cases neuritis is prominent. Jendrassik and Marie¹⁹ also speak of infectious diseases as a cause of hemiplegia. Cotard recognized this factor at a very early date. Osler has noted a connection with infectious diseases in sixteen of his cases. (l. c.) Freud and Rie²⁰ were able to refer five of their thirty-five cases of infantile hemiplegia to this origin. Moraga²¹ describes hemiplegia at the age of twenty-two months after measles, and Oppenheim²² at the age of eight years after whooping cough. Numerous other cases are found in the literature, but the tendency, especially among French writers (Sachs), has been to ascribe too many cases to infectious diseases.

Marie²¹ thinks that hemiplegia beginning after the age of nine should not be called infantile. In forty cases of infantile hemiplegia examined by Sachs²³ the causes were atrophy, sclerosis and cysts; in two cases porencephaly was found, in twenty three hemorrhage, in seven embolism, in five thrombosis, in one agenesis, and in another tubercle. Intracerebral hemorrhage in the young is more common than is generally supposed (Sachs). Hereditary syphilis does not seem to be of much importance (Sachs, Osler). The remarkable case

¹⁶Dercum, *Nervous Diseases*, p. 509.

¹⁷Imogene Bassette, *Journal of Nervous and Mental Disease*, 1892.

¹⁸Jendrassik and Marie, *Archives de Physiologie*, 1885, p. 51.

¹⁹Freud and Rie, *Klinische Studie über die halbseitige Cerebral-lähmung der Kinder*.

²⁰Moraga, abstract in *Neurologisches Centralblatt*, No. 11, 1896.

²¹Oppenheim, *Deutsche Med. Wochenschrift*, 1896, No. 17.

²²Marie, *Dictionnaire Encyclopédique des Sciences Médicales*, 1888.

²³Sachs, *Sammlung klinischer Vorträge*, Inner. Med., 1-29, p. 470.

of aneurism of a branch of the anterior cerebral artery in a boy of six years, recorded by Osler²², is well known. Dejerine²³ reports three cases of infantile hemiplegia with autopsy. In two of these the lesions were due to hemorrhage; the third was probably due to ischæmia. One case occurred in convalescence from scarlet fever. Starr²⁴ gives his opinion very firmly in favor of cerebral hemorrhage as the cause of these birth palsies, and Osler favors embolism (l. c.) Schultze²⁵ has recently reminded us that hemorrhage during birth may occur in the spinal cord, and not necessarily in the brain. The frequency



FIG. II.—Hyperextension of the big toe in infantile hemiplegia.

of this is doubtless considerably less than that of cerebral hemorrhage. Pfeiffer²⁶ expresses this view. He examined five cases; in four of these the forceps had been employed, in one version had been practiced. Only once did he find macroscopically the signs of extensive hemorrhage in the cord, and he concluded that hæmatomyelia from dystocia should not be regarded as com-

²²Osler, *Canada Medical and Surgical Journal*, 1886 (quoted by Sachs).

²³Dejerine, *Archives de Physiologie*, 1891, p. 661.

²⁴Starr, *Medical Record*, 1892

²⁵Schultze, " *Deutsche Zeitschrift für Nervenheilkunde*, VIII., Nos. 1 and 2.

²⁶Pfeiffer, *Centralblatt für Allgemeine Pathologie, u. Pathologische Anatomie*, Sept., 1896.

mon as the first investigations seemed to indicate. Nevertheless, one out of five cases seems to us a large percentage, and further examination will probably show that it is too great. Peterson,² referring to the fact that Herbert R. Spencer, in a hundred and thirty stillborn children, found hemorrhages into the spinal canal and cord in thirty cases, says he cannot but believe that some, though a very small percentage, of the cases of paraplegia especially, and perhaps diplegia, may be due to cord lesions at birth after all, and not to cerebral lesions.

In some of our cases the hemiplegia was first noticed after convulsions at the age of four, five or six years. It is not improbable that a lesion may have occurred during birth, or within the intrauterine period, and that the sclerosis may have taken a few years to reach a stage sufficient to cause convulsions and hemiplegia. The case we present in this paper, with a photograph of the brain (Fig. 6.), is in evidence of the long duration of sclerosis without epilepsy. In another case, which was congenital, the epilepsy first developed at the age of twelve.

We hardly think it worth while to note the statements in regard to difficult labor, inasmuch as we are unable to obtain reliable answers from the parents. These blanks have not been filled out by the attending physicians, and many women are inclined to regard a normal labor as difficult. We do not mean that this question is of little importance in infantile paralysis, for Little, Gowers, and others in England; Sarah McNutt, Sinkler, etc., in this country, have taught us differently, and yet Lovett says: "It seems reasonable to conclude that the influence of difficult labor in producing cerebral paralysis must have been greatly overestimated." He also quotes Langdon Down as saying: "I found that among the great number of feeble-minded children about whom I could get thoroughly reliable accounts, in only three per cent. had the forceps or any other instrument been used."

It is also not worth while to enter into a discussion of Strümpell's theory in regard to acute infantile encephalitis, as we have no facts to bring forward for or against this view.

²Peterson, *An American Text-Book of the Diseases of Children*. Edited by Louis Starr. p. 657.

The French authors especially have made very prominent the peculiar condition of the foot in Friedreich's disease, of which one of the chief features is the hyperextension of the big toe, and to a less degree of the others. The first phalanx is over-extended, while the second is flexed. This hyperextension may be excessive. The position of equino-varus is usually present, but is not essential, according to Brissaud¹⁹. Two excellent pictures of the condition are given by this author. Marie²⁰ also describes this alteration of the foot, and regards it as



FIG. III.—Ulnar Oligodactyly.

a relatively early sign of Friedreich's disease. In certain families, in which several members are afflicted with this malady, the hyperextension of the big toe indicates the beginning of the process. Auscher²¹ also presents a picture of the deformity in a case of Friedreich's disease. Higier²² has reported four remarkable cases, which he considers atypical forms of cerebral diplegia. In all four

¹⁹Brissaud, *Leçons, sur les Maladies Nerveuses*, pp. 57, 58.

²⁰Marie, *Leçons, sur les Maladies de la Moelle*, p. 391.

²¹Auscher, *Archives de Physiologie*, 1893, p. 342.

²²Higier, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. 9, 1896.

this hyperextension of the big toe is well represented in the pictures. Two of our cases of hemiplegia present this deformity (see Fig. 2). We are not aware that this has been mentioned in studies on hemiplegia. We would imagine from the picture that it was present in Sachs' case of congenital diplegia known as the "Frog Girl." This hyperextension, however, can not be considered as pathognomonic of Friedreich's disease.

Féré³⁴ has devoted much attention to the deformities in infantile hemiplegia. He has described a condition which he calls ulnar oligodactyly ("oligodactylie cubitale"), which is seen chiefly in the ring finger. The little finger often takes part in the anomaly. Occasionally, in normal persons, the ring finger is shorter than the index, but in forty two cases of infantile hemiplegia with atrophy of the hand Féré found this condition thirty-six times. We have observed this shortness of the ring finger in nine cases (see Fig. 3). In four cases athetoid movements have been noted.

Occasionally the cerebral form of hemiplegia may be difficult to distinguish from the spinal. The following case is an example of hypotonia in cerebral hemiplegia.

Case No. 1.—The paralysis is in the right upper and lower limbs. Voluntary movement at the right shoulder and elbow is restricted and awkward. In the fingers of the right hand the voluntary movement is very imperfect, and yet the right arm can be raised passively above the head as high as the non-paralyzed limb, the forearm can be flexed passively with great ease. There is no resistance in the right shoulder and elbow to passive movement. There is a slight tendency to flexor contracture at the wrist, which can be easily and fully overcome. There is also a slight tendency to flexor contracture in the fingers. The right upper extremity is shorter than the left, and the difference is very noticeable in the hand. The musculature of the entire limb is less developed than that of the left. In the right lower limb passive movement reveals also a condition of moderate hypotonia. The foot is in the position of slight talipes equinus, but can be easily extended, and in walking the entire foot is placed upon the ground. Active movement is free at all joints of the lower limb, but the right knee is raised a

³³Sachs, *Nervous Diseases of Children*, p. 531.

³⁴Féré, *Revue de Médecine*, Feb., 1896, p. 115.

little higher than is normal in walking, as the toes of the right foot have a tendency to cling to the ground. The patient has on this side a very slight degree of the "stepper gait," as it is called by Charcot. The entire right lower limb is shorter than the left, but the difference is in the leg and foot; the thigh is about normal in length. The musculature of the entire limb is smaller en masse. The patella reflex is present on both sides, but is not exaggerated. The reflexes of the right upper limb are not increased.

On account of this condition of hypotonia, which



FIG. IV.—Hypotonia of fingers in the paralyzed hand.

is very perceptible, though not excessive, and seems to be more than imperfect development of the muscles would account for, we have hesitated at first in excluding poliomyelitis. The preservation of the reflexes, the slight tendency to contracture and the diminution in size of the hand and foot, parts which are supposed to be more exclusively under the control of one side of the brain than the rest of the limbs, as the generally accepted theory teaches, and the very feeble mental condition, make the case undoubtedly of cerebral origin. We note also the considerable diminution in the right thorax, amounting to an inch and a quarter, although the right arm and thigh are not much smaller, the differences being chiefly in the forearm and hand, leg and foot.

Sachs and Peterson²⁴ report two cases of cerebral palsy in which the element of spasticity was entirely wanting, although the cases were undoubtedly of cerebral origin, the proof of which was furnished by the post-mortem examination in one of the two cases. Osler also reports three cases in which the arm was flaccid (l. c.) Usually, as in the case we report, there are sufficient signs to make the cerebral nature of the affection evident, even where spasticity is absent. In another paper Sachs²⁵ makes the following statements: "I can safely say that of the two hundred and five cases we have seen, doubt as to the spinal or cerebral origin was entertained in but a single instance." "Since the foregoing was written, I have seen one case, and the only one, in which there was evidence of a spinal and a cerebral lesion."

Case No. 3 is an example of retardation of growth in the extremities of the paralyzed limbs, which we have noticed in so many of these cases. The lower limbs are about the same length, and yet the foot of the paralyzed side is much smaller than that of the other side. In the upper limbs the difference is slight in the length of the two forearms, but in the hands this is much greater. There seems to be no explanation for this if we reject the theory that the extremities of the limbs are more exclusively innervated from one side of the brain. It is well known that motion is usually more impaired in hemiplegia at the extremity of the limb.

We may, if we wish, believe that the degeneration of the fibres in the crossed pyramidal tract of the "sound" side in a case of hemiplegia begins at the motor decussation, and Rothmann²⁶ has recently explained this as the result of pressure on the fibres coming from the normal pyramid by the degenerating fibres from the affected pyramid. In the early stages of degeneration there is swelling of the fibres and pressure is exerted. This is a little different from the theory of Hallopeau.²⁷ According to the view of this writer, the degeneration of the pyramidal tract of the "sound" side begins also at the decussation, but the cause is transmitted inflammation from the fibres of the degenerated pyramid to those of

²⁴Sachs and Peterson, *Journal of Nervous and Mental Disease*, 1890.

²⁵Sachs, *New York Medical Journal*, 1891, p. 503.

²⁶Rothmann, *Neurologisches Centralblatt*, Nos. 11 and 12, 1896.

²⁷Hallopeau, *Archives generales de Médecine*, Vol. 11, 1871.
(Quoted by Rothmann.)

the sound. This theory of the origin of the degeneration of the fibres of the crossed pyramidal tract of the "sound" side at the decussation does not explain such a condition as is presented in these cases. We can not understand why the extremities of the limbs are so often more involved on the paralyzed side unless we believe that, while those portions of the limbs nearest the trunk have a bilateral, the hand and foot have a more exclusively unilateral cerebral innervation. If the degeneration of the crossed pyramidal tract, which has been found on the "sound" side, does not represent fibres which come from the cerebral hemisphere on the same side as the lesion, then we have little or no anatomical proof that such fibres really exist, and the innervation of each limb



FIG. V.—Unilateral arrest of development of the face in infantile hemiplegia.

from both sides of the cerebrum must remain merely as a theory. Rothmann, however, does not stand on the side of the majority of investigators. Dejerine and Thomas" state they have seen degenerated fibres passing from one pyramid to the crossed pyramidal tract of the same side. Rothmann describes similar fibres, but be-

"Dejerine and Thomas, Archives de Physiologie, April, 1896,

believes that their course excludes the possibility of their origin in the pyramid of the same side.

It is well known that the foot can be trained to make almost all the movements of the hand. We can recall an artist in the art gallery of Antwerp who, being deprived of both arms, made an excellent copy of one of the paintings by means of his feet—mixed his colors and picked up his brushes and palette by means of his toes alone. It seems easy of comprehension that for these highly specialized movements of hand and foot special cells on one side of the brain should be set apart, while for those movements of both sides of the body which usually occur together both cerebral hemispheres should be employed. How else shall we explain associated movements in paralyzed limbs?

In examining spinal cords from cases of hemiplegia by the method of Marchi we have found several times both crossed pyramidal tracts degenerated. This is by no means a recent discovery. It must be remembered that there is no truly sound side in hemiplegia.

The fingers and thumb in this case (No. 3) are in flexor contracture. In many of these cases of contracture of the wrist joint the fingers are in extension.

The view of Rothmann that the degeneration of the crossed pyramidal tract on the so-called sound side begins at the motor decussation explains the increased reflex of the "sound" side as well as does the theory that fibres extend from the cortex to the lateral column of the same side of the cord.

Cases Nos. 4 and 5 are examples of the abnormal flaccidity (hypotonia) of the fingers on the paralyzed side, seen in at least six of these children. The fingers can be bent backwards almost at a right angle (see Fig. 4).

We have noticed this flaccidity of the fingers even when the wrist was in flexor contracture.

In case 5 the upper limb on the paralyzed side is shorter than on the "sound" side, and the thorax is three-quarters of an inch smaller. There is impairment of motion at the shoulder but none at the hip, as is seen in many of these cases. This indicates, probably, that the two sides of the brain are concerned in the movements of the hip joint more than in those of the shoulder. Flexion of the fingers is much better than extension, as is usually the case, and abduction and adduction of the fingers are most interfered with.

Case No. 6 presents latent contracture of the wrist joint on the paralyzed side; any manipulation of this joint causes increased flexion of the hand. If the hand is allowed to rest quietly on the examiner's knee it remains partially extended. It is a question if it would be wise in a case like this to resort to passive movement, as permanent contracture might develop. There are athetoid movements in the paralyzed hand.

Exaggeration of the reflexes may remain after motor power has been almost fully restored, as is seen in case No. 7. The patella reflex on the paretic side is exaggerated, but the gait is almost normal. This patient was born in normal labor. At the age of two she had a convulsion, which was followed by loss of sight, hearing and speech. Aphasia has entirely disappeared. This was a case of *left* hemiplegia. There is an uncertain history of brain disease when the child was eight months of age, which was supposed to have been the cause of the hemiplegia.

Case No. 13 has a very interesting feature. The patient has left hemiplegia, with paralysis of the muscles innervated by the lower branch of the seventh nerve, and the left side of the face is smaller than the right (see Fig. 5). It is true Féré⁴ remarks that the face usually participates in the hemiatrophy of infantile hemiplegia, and is more or less asymmetrical. This is a somewhat surprising statement. We have only found one case of arrested development in which the face was involved.

Case No. 16, contrary to the customary condition, presents greater contracture at the elbow joint than at any part of the paralyzed upper limb.

Case No. 18 is one of the most interesting we have examined, and of practical importance. Wiener⁴ has recently published a case of progressive muscular dystrophy which was considerably improved by methodic gymnastics. Zabłudowski⁴ has reported a case of Friedreich's ataxia which was much benefited by massage employed for four weeks. Nature, by means of athetosis teaches us how continued exercise of the paretic limbs develops the musculature. Contracture and athetosis seldom are present together in severe form. Sachs⁴ recommends massage and electrical treatment in

⁴Féré, *Les Épilepsies et les Épileptiques*, p. 31, also *Revue de Médecine*, No. 2, 1896.

every case of cerebral paralysis, to prevent contracture. From this statement it would seem that there is no fear of developing the latent tendency to contracture, what the French call the "imminence" or "opportunité de contracture." Our case No. 18 employed such exercise of her own accord. These cases are encouraging in view of the hopelessness usually felt toward the possibility of improvement in such conditions. The patient, A. K., is very intelligent for her station in life. The left hemiplegia is supposed to have been congenital. The patella reflex on the two sides is equally increased, and ankle clonus is present on both sides. Reflexes of both upper extremities are exaggerated, though more on the paralyzed side. The patient can raise her left arm at the shoulder almost as high, and can draw it backward almost as far as the right. She can almost fully extend the left forearm, but cannot extend the hand beyond the plane of the forearm. She has considerable power of adduction and abduction in the fingers, and these movements we have found in all the other cases the most interfered with. She can flex the fingers, but has little power of flexion at the wrist, and opposition of thumb with fingers is very imperfect. The left upper limb is much shorter than the right, and the difference is more noticeable in the forearm and hand. The musculature of the left upper limb is somewhat less developed than that of the right, but the difference is not as great as is usually found in these cases, probably on account of the exercise of the muscles. The left hand and fingers are very much smaller. The grasp of the left hand is rather feeble. In the lower extremities the thighs cannot be flexed on the abdomen as much as in most of these cases, although the right can be flexed a little more than the left. This in connection with the equal exaggeration of the patella reflex on the two sides would seem to indicate that in this case the pyramidal tract on either side supplies the lower limbs more nearly equally. There is no power of flexion or extension at the left ankle joint; the toes of the left foot cannot be moved. The left lower limb is much shorter than the right, and the difference is greater below the knee. The musculature is also less developed. In walking the toes of the left

⁴¹Wiener, *Neurologisches Centralblatt*, May, 1896. *The American Journal of the Medical Sciences*, Oct., 1896.

⁴²Zabludowski, *Berliner Klinische Wochenschrift*, No. 34, 1896.

⁴³Sachs, *Volkmann's Sammlung klinischer Vorträge*.

foot touch the ground, and the heel is held elevated. The left side of the thorax is three-quarters of an inch smaller than the right.

In view of the exaggeration of the reflexes, passive movements might have been thought inadvisable, but the result has not justified such a fear. There is contracture only at the ankle and wrist. There are cases of infantile hemiplegia which improve spontaneously, and the question might arise if this is not one of these cases. The exaggeration of the patella reflex, the decided ankle clonus, and the increase of the reflexes in the upper limbs on both sides, the loss of power at the wrist and ankle, the

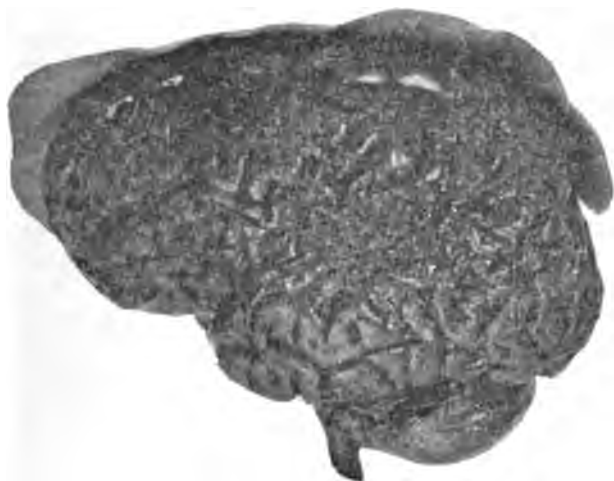


FIG. VI.—Sclerosis in the distribution of the Sylvian artery. From a case of infantile hemiplegia.

retarded growth of both left limbs and of the left side of the thorax, make it doubtful if improvement would have been so great if means had not been used. The patient, who is intelligent and can read and write, informs us that up to the age of nine years the power in the left upper limb was much less than at present. At this period of life she began systematically to use the left upper limb in order "to see how much she could carry." She would pick up a pail of water and carry it in her left hand, and in this way power became gradually greater in the arm. This was her own idea. She would raise

the left arm at the shoulder with the right hand, and would forcibly extend the left hand.

An interesting fact stated in the history is that the mother of this patient was also paralyzed in the left side. While we do not know that the mother was paralyzed previous to the birth of this child, we learn that she lived but fourteen months after the child was born. It looks very much as though the condition were hereditary in the daughter. Such cases have been recorded (Oppenheim).

Case No. 17 has a peculiar condition of the hand on the paralyzed side. The contracture at the wrist is extreme, the hand is so much flexed that it almost touches the front of the forearm. the fingers are also flexed, and the hand can only be extended passively far enough to form a right angle with the forearm. According to the statement of the patient, who is intelligent, and to the observations of those about him, every morning the hand is less contracted, and is only slightly flexed at the wrist, the fingers are then extended, and he has some power of movement. This condition lasts only a short time, and very soon the hand and fingers are again extremely flexed. The increased contracture as the day passes is probably due to summation of irritation brought about by constant demands on the reflex action. We can recall in the clinic of Professor Dejerine, at the Salpêtrière, a patient with myelitis, in whom the lower limbs showed this alternate condition of flexion and extension.

Case No. 30 exhibits contracture in both legs and one arm. The muscles of the left shoulder are much contracted and offer great resistance to passive movements in all directions; the forearm can only be extended passively to an angle of about 115° with the arm, the hand forms a right angle with the forearm, and cannot be moved by the patient in any direction. There is slight power of flexion in the fingers. The fingers can be moved passively in all directions.

Both lower extremities are equally rigid, abduction is lessened, flexion of the right thigh on the pelvis and of the right leg at the knee is not possible beyond a right angle. Active movement in the left lower limb is only a little less than in the right. The two legs are of equal length. The entire left upper extremity is much smaller than the right. The two sides of the thorax are exactly equal.

In walking the left limb is dragged from the hip and

the knee and ankle are held rigid. The right knee is kept partly flexed. The patella reflex is exaggerated on both sides, but more on the left. Epileptic attacks occur at irregular intervals. Nothing is known of the early history of the patient. This condition may be due entirely to an unilateral lesion, or it may be that the lesion has extended over the superior part of the hemispheres and involved the leg centre of the opposite side.

Case No. 31 has a very interesting tremor on the paralyzed side. There are occasional involuntary twitchings of the left side of the jaw. The paralyzed arm is in a state of constant tremor, which is most noticeable in the hand. The same condition is observed in the lower



FIG. VII.—Porencephaly from a case of infantile hemiplegia.

limb. At times the tremor may be called fascicular, again it is so strong that the leg moves considerably. These post-hemiplegic movements are well known since Weir Mitchell long ago called attention to them.

We present a photograph (see Fig. 6), kindly taken for us by Dr. Robert Formad, of a very interesting brain. We do not enter into a detailed description, as we are now studying the case by means of serial microscopic sections.

The patient was paralyzed on the right side "from a fright" in early infancy. Epilepsy did not develop until the age of ten. It is surprising that convulsions were

not observed sooner, in view of the extensive sclerosis. The boy's vocabulary was very limited. It will be noticed that Broca's region was involved. Impairment of the mental faculties was present. Until the age of thirteen the boy's mother states that he was able to walk. At this period progression became impossible. As the right hemisphere presented no lesion at the autopsy, it is fair to assume that the weakness of the left lower limb was due to the sclerosis in the left hemisphere, and the case is in evidence of bilateral innervation from each hemisphere. The fact that the sclerotic area was limited to the distribution of the middle cerebral artery makes the theory of extensive progression of the process doubtful. Unfortunately we have not been able to examine the spinal cord, but there is no reason to suppose that there was a spinal lesion in addition to the cerebral. There can hardly be a doubt that the cause of the lesion in this case was an embolus or thrombus, more probably the former. Only the area nourished by the Sylvian artery was sclerotic. The upper part of the Rolandic region, which receives branches from the anterior cerebral artery, almost all the frontal, the whole of the occipital lobe, and the second and third temporal convolutions are normal. These parts do not receive their blood supply from the middle cerebral artery.

The clinical history of the interesting case of porencephaly we present (see Fig. 7) is very meagre. The patient's mental condition was of low grade. The right side of the body was imperfectly developed and was paralyzed. The statement is also made that the patient never used his right hand.

It will be noticed that the cavity involves Broca's area, and yet this boy is said to have "sung sweetly." He could not, therefore, have been aphasic, at least in singing.

More fibres were cut or remained undeveloped than a superficial examination would give reason to suspect, for we find by a microscopic study of the oblongata that the left pyramid is absent while the right is unusually large. The gyri do not end abruptly at the borders of the cavity, but with the pia pass into its depths. Later we will probably have something more to say of this interesting specimen.

SYNOPSIS OF SYMPTOMS OBSERVED IN 33 CASES OF INFANTILE HEMIPLEGIA.

Hyperextension of the big toe on the paralyzed side ; number of cases, 2.

Aphasia in right hemiplegia, without disturbance of intellect, only in one case, and this patient is deaf as well as dumb.

Ability to write in the eight cases of right hemiplegia examined is present in 5. One patient can print, one can copy script as script ; one can not write at all. Education must be thought of in this connection.

Age of Onset of Hemiplegia.	Age at Time Examined.
18 mos.	20 yrs.
Congenital	not known.
Not known.	14 yrs.
Not known	18 yrs.
6 yrs	18 yrs.
Congenital	12 yrs.
Infancy.	18 yrs.
3 weeks	10 yrs.
Congenital	20 yrs.
2½ yrs	19 yrs.
Congenital	31 yrs.
8 mos.	14 yrs.
Not known.	18 yrs.
Not known	23 yrs.
10 yrs	18 yrs.
16 mos.	19 yrs.
Infancy	not known.
Congenital	22 yrs.
Congenital	9 yrs.
Not known	about 13 yrs.
Congenital	14 yrs.
Not known	20 yrs.
Congenital	12 yrs.
Not known	20 yrs.
2 yrs.	20 yrs.
Not known	33 yrs.

	No. of Cases.
Ulnar oligodactyly	9
Athetosis	4
Facial paralysis or paresis	10
Unilateral arrest of facial development	1
Epilepsy	22
Convulsions only in infancy	5
Sensation in all cases normal.	

Etiology.	No. of cases.
"Brain fever,"	1
Small pox	1
"Catarrhal fever,"	1
"Confusion of brain,"	1
"Congenital tubercular disease,"	1
"Congestive chill,"	1
Measles or scarlet fever	1
Aura ascertained	6
Diminution in size of thorax on the hemiplegic side	16
Retarded growth in the paralyzed limbs	31
Presumable heredity	1
Weakness of intellect to a notable degree	13
Hypotonia of fingers	6
Right hemiplegia	17
Left hemiplegia	16
Boys	10
Girls	23
Contractures of the paralyzed limbs	31
Unilateral paralysis of the tongue	4
Paralysis of the muscles supplied by the fifth nerve not observed.	
Unilateral atrophy of the tongue	1

In addition to these cases from the Pennsylvania Training School for Feeble-Minded Children, we report a case of congenital chorea studied at the New Jersey Training School for Feeble-Minded Children.

The boy was born at full term with the use of forceps. Deficient animation was noticed at birth. As a babe he was rather sickly, and did not begin to walk until nearly three years of age. His mother states that his condition was congenital. At present he is a well developed lad. The tendon reflexes of the lower limbs are greatly exaggerated. A tap on the patella tendon throws the limb into a rigid state. Patella clonus and ankle clonus are present. There is a tendency to pes equino-varus. What might be called fascicular chorea (Klippel and Durante) is present in the entire body, even the face and tongue are involved, and when the eyes are forcibly closed the involuntary fascicular movements of the face and the tremor of the head are increased. The tremor of the hands is noticed especially when the arms are extended, but it is not as severe as in the lower limbs. The speech is very indistinct and is like that of ordinary chorea. The pressure of the hands is strong and apparently equal on the two sides, and no weakness can

be detected in the lower limbs. Sensation for touch and pain is normal. The gait is incoördinate, but not spastic. He complains of some weakness in the left arm and leg. The muscles supplied by the lower branch of the left seventh nerve are paretic.

The case should be classed under hemiplegia. The greatly increased tendon reflexes, the subjective weakness on the left side of the body, the paresis of the left facial muscles all point in this direction. There is in this case in all probability some cerebral lesion irritating but not destroying the motor centres.

Critical Digest.

THE PATHOLOGY OF SYRINGOMYELIA.

By M. ALLEN STARR.

There is at present little discussion regarding the symptomatology or diagnosis of syringomyelia. The disease is readily recognized, and many cases have been recorded in which the diagnosis has been confirmed by post mortem examination.¹ The extensive literature of the subject is to be found in Schlesinger's² and Dimitroff's³ monographs, where more than five hundred references to published articles are given.

The chief interest in the disease now centres about its pathology, regarding which many diverse views prevail. This diversity of opinion is traced by Hoffmann⁴ to the fact that the cases which have been examined have rarely if ever come to a fatal end in the early stages of the disease, and hence the origin of the process has to be deduced from the terminal appearances. These appearances are subject to individual interpretation, and each pathologist in turn has tried to bring all cases into conformity with his own observations upon his own particular cases. The various views which have been brought forward to explain the existence of cavities within the spinal cord are, in the order of their proposal, as follows:—

a. The view of Virchow, who ascribed all such cavities to a distension of the central canal. This view was modified by Leyden, who considered the condition a congenital hydromyelus. These views are now modified by their authors.

b. Simon first distinguished sharply between hydromyelus and syringomyelia, and pointed out the existence in the latter of gliomatous tissue and its destruction.

c. Schultze then urged the view that prior to the formation of any cavity there was always a central gliomatosis, a proliferation of glia cells, which, though usually beginning near the central canal, may begin anywhere in the cord.

d. Hallopeau later proposed the theory that the condition was really a chronic inflammation beginning in the epithelium of the central canal or in the periependymal cells, and considered the process of the nature of sclerosis.

e. Joffroy, like Schultze, denied any necessary relation between the pathological process and the epithelium of the central canal, but considered that an inflammatory change might begin in any part, leading finally to disintegration, a true "myelite cavitaire."

f. Langhaus then brought forward an hypothesis, which Kronthal subsequently supported and which Müller and Medin¹ have recently urged, that an obstruction to the venous or lymphatic return flow from the cord may lead to accumulation of fluid in the central canal, distension of the cavity of the canal, pressure on, and thus disintegration of adjacent gray matter, with glia formation.

g. Lastly, Van Gieson, from a study of a number of cases, has shown that a long cavity may be left after the absorption of the clot formed by a perforating hemorrhage in the cord; that such a cavity is surrounded by a zone of neuroglia, thus simulating true syringomyelia. Dana, while admitting the possibility of perforating hemorrhage confined to the gray matter of the cord, considers that a true necrotic process is in some cases the primary cause of such cavities as Van Gieson describes.

When the various views regarding the exact origin of cavities in the cord are tabulated, it appears that the following views have a well-established basis in pathological observation:—

Cavities may be produced by

I. A dilation of the central canal.

a. Congenital from maldevelopment.

b. Acquired as hydrocephalus.

II. A maldevelopment of the central canal.

a. With supernumerary canal or double canal.

b. With imperfect closure of the posterior septum.

These conditions (one or more) are thought by many authors to be present as an original predisposing cause in every case of syringomyelia. It is to be remembered that the central canal is surrounded by a large number of small epiblastic cells, whence the neuroglia develops in a normal cord, and that the formation of new neuroglia tissue is a part of the lesion in syringomyelia, and that the situation of the cavity or of the neuroglia prolif.

eration. points to some necessary relation between these epiblastic elements and the new tissue.

III. A primary proliferation of the neuroglia tissue of the cord, without any maldevelopment, and a gradually advancing disintegration of the new tissue at its centre, may lead to the formation of a cavity with walls of thick neuroglia. This is termed gliomatosis.

a. This may be a single hyperplasia from unknown causes.

b. It may be inflammatory, produced by trauma, by irritation of poisons, or by infection.

c. It may be a true tumor formation.

There may be a relation between the existence of numerous embryonal cells, capable of development into neuroglia, near the central canal, and the process of gliomatosis, as it seems reasonable that such a process should begin in these cells when from any cause it is set up. But authors dispute this relation, and some hold that the proliferation may begin anywhere in the cord, in normal neuroglia tissue. In this connection the views of Weigert regarding neuroglia may be cited, and his opinion concerning the lesion in syringomyelia.

Weigert (*Die Neuroglia*, 1896) shows that new formations of neuroglia may be either cellular in structure or fibrous in structure. If cellular, the new formation is a true glioma, such as is found in tumors of the brain or spinal cord, and in such a glioma fibres are few. Weigert calls attention to the fact that this is not the structure of the glia tissue about the cavity of syringomyelia, but that the neuroglia found around this cavity consists almost exclusively of glia fibres with few cells, and that these fibres, though extending in all directions, are chiefly vertical in their course. Miura¹ also has shown the sharp contrast between ordinary glioma, even glioma containing a cavity, and the gliomatous condition of the cord in syringomyelia. Weigert holds that the neuroglia is merely a substance produced by nature to take the place of nerve tissue which has been destroyed, and that its proliferation is always an indication that the nerve tissue has primarily disintegrated. Some destruction of nerve tissue would therefore, according to his idea, precede the formation of gliomatous tissue, and hence he discards wholly the theory of syringomyelia to which the term spinal gliosis has been applied. Weigert says "many authors believe that the essential lesion in syringomyelia is the formation of a tumor followed by soften-

ing and the formation of a cavity. It is admitted that there is a growth of neuroglia of the typical fibre-type about the cavity. But this fibre mass, devoid of cells, does not resemble a glioma, and there is no reason to believe from the mere presence of neuroglia that the cavity is not a congenital or acquired abnormality of the central canal. There is a thick cluster of neuroglia fibres normally about the canal. By the pressure in this enlarged canal the nervous tissue may be destroyed and hence a growth of neuroglia fostered. If the pressure increases the neuroglia may also be destroyed, and in its place about the cavity a hyaline formless mass may remain. The gliosis is not the essential feature; it is only a secondary result." (*Centralbl. f. Path. u. Path. Anat.*, Vol. I., p. 736 and *Neuroglia*, p. 101.)

On the other hand, many authors have noticed the great proliferation of neuroglia cells in the segments of the spinal cord above and below the limits of the cavity and have considered this a proof that the proliferation of these cells was a necessary precursor to the formation of the cavity, a view exactly opposed to that of Weigert.

Some authors (Miura) have sought to contrast the true glioma sometimes found in the cord with the gliomatosis of syringomyelia, but Turner and Mackintosh¹ have published cases in which every possible transition between these conditions is evident. It is true that sometimes a glioma, or a sarcoma, or a gliosarcoma has been found within the cavity (Van Gieson) in cases of syringomyelia, and Hoffmann¹ describes these cases as a special class. But it is not always possible to determine whether the cavity or the tumor is the primary condition or whether both were varieties of the same pathological process going on in different ways at different levels of the cord. Miura has described a case in which there was a diffuse glia proliferation in the gray and white matter from the mid-cervical to the upper lumbar region without any cavity formation whatever, but with here and there pale foci which might be considered areas of beginning degeneration. This he considers a true tumor formation, not a chronic inflammatory neuroglia proliferation and without any relation to the central canal. In Turner's cases the tumor and the canal were not at the same level, but ran into one another, and here, also, there was no evident relation between the canal and the pathological processes.

IV. A softening and disintegration of cord tissue

may be followed by the formation of a cavity with a wall of new neuroglia or without any wall. Such cavities are thought to be due to disease in the walls of the spinal arteries. Müller and Medin⁶ have seen a cavity in the gray matter of the cord with walls of normal nerve tissue with no signs of inflammatory disease or of proliferation in the glia, the size of the cavity corresponding in situation to the degree of endarteritis in the spinal vessels, and having no definite relation to the central canal. Wieting⁷ has described a cord containing numerous cavities, due entirely to the low nutrition of the nerve tissues and consequent necrosis from disease of the spinal arteries in connection with meningomyelitis. The cavities were of various shapes, and their walls contained disintegrated neuroglia cells and many blood vessels. Here and there the wall of the cavity was made of thick neuroglia with many fibres and numerous cells. At other places a hyaline infiltration was seen in the wall of the cavity and in the surrounding tissue, which was considered a terminal phase of glia disintegration. In this cord many collections of neuroglia cells in foci and stripes along the blood vessels were seen. There was no relation between the situation of these cavities and the central canal. The capillary vessels were increased in the foci of neuroglia and in the walls of the cavities forming a tangled mass with thick sclerotic walls and sometimes with obliterated lumen. These appearances do not, however, resemble at all closely the appearances characteristic of syringomyelia, and Hoffmann rightly discards such cases of necrotic spinal cavities from the classes of pathological varieties of syringomyelia. It has been thought by Kronthal that lymphstasis within the cord and consequent necrosis produced by transverse compression of the cord may lead to the formation of cavities which may or may not communicate with the central canal, but this theory is doubted since compression of the cord by tumors or after Pott's disease is not found to cause cavities, and such cavities as he describes certainly differ from those found in syringomyelia in the absence of a wall.

V. Lastly, there is not wanting a theory of the disease which would trace it to an infection of the cord by a bacillus. The resemblance between Morvan's disease and syringomyelia has been noticed by all writers, and their identity has at last been established by the fact that in more than a dozen cases of Morvan's disease

the lesion of syringomelia has been found post mortem in the spinal cord.¹ It is true that in Morvan's disease an infection of the peripheral nerves has also been found to exist in some cases, but it remains an open question whether this peripheral neuritis is not an expression of a disturbance of trophic action due to the lesion in the spinal cord. Hoffmann considers that the cases of Morvan's disease are merely a variety of syringomyelia and maintains that there are no clinical differences between the affections. But the similarity between Morvan's disease and lepra anæsthetica has been noticed by more than one author, Zambaco having especially called attention to the resemblance. It is known that lepra anæsthetica is produced by a bacillus, and many writers upon Morvan's disease have maintained that the fact that this disease develops, particularly along the coast of Brittany, among fishermen, and in certain localities rather than in others, offers a positive proof that it has some extraneous origin, probably of the nature of an infection. In a very exhaustive article on the relation between syringomyelia, lepra and Morvan's disease, Prus¹¹ reaches the conclusion that these three diseases may be varieties of the same general condition, Morvan's disease being the least intense, lepra anæsthetica the most severe. Lepra depends upon the existence of a bacillus which is now well recognized. Strauss has found this bacillus under the skin in cases of Morvan's disease which he examined in Brittany, and this has been confirmed by Moniez and Roux. Prus, therefore, has proposed the hypothesis that the lepra bacilli, after penetrating the skin, may set up a degeneration of the nerve fibres, causing the well-known symptoms of Morvan's disease; that then they may migrate along the course of the nerves, leading to changes in the nerve trunks and thus ascending the nerves to the spinal cord, where they may produce gliomatosis, with its subsequent destruction, and hence the appearance of syringomyelia. Prus does not affirm that all cases of syringomyelia originate in this manner, but believes that a certain number may be thus explained. On the other hand, it is to be remembered that Schultze draws a sharp line between lepra and syringomyelia and denies the existence of any spinal lesions in lepra. And other authors who have examined the spinal cord in lepra have found the bacilli of the disease in the cord and in the cerebro spinal fluid without finding any condition of gliosis or any degeneration of the nerve tissues. The identity of lepra

and syringomyelia is by no means established, and therefore this theory of an infective cause for syringomyelia cannot be as yet accepted.

The conclusion to be drawn from this review of various theories is that cavities develop from many causes, and that the lesion of syringomyelia is not of uniform causation.

¹Prof. J. Hoffmann. Zur Lehre von der Syringomyelie. Deut. Zeit. f. Nervenheilkunde III., 1893.

²Prof. A. Chiari. Ueber die Pathogenese der sogenannten Syringomyelie. Zeit. f. Heilkunde, IX., 307.

³Joffroy et Achard. Syringomyelie et Maladie de Morvan. Archiv. de Med. Experimentale et d'Anatomie pathologique. III., annee 1.

⁴A. Miura. Ueber Gliom des Rückenmarkes und Syringomyelie. Ziegler's Beiträge zur Path. Anat., XI, 91, 1892.

⁵F. Wieting. Ueber einen Fall von meningo-myelitis chronica mit Syringomyelie. Ziegler's Beiträge zur Path. Anat., XIX., 215, 1896.

⁶Mueller und Mendin. Zur Kenntniss der Syringomyelie, Zeitschr. f. Klin. Med., XXVIII., 128, 1895.

⁷H. Schlesinger. Die Syringomyelie. Leipzig, 1895.

⁸S. Dimitroff. Über Syringomyelie. Archiv. für Psych., XVIII., 1896.

⁹Turner and Mackintosh. Three cases of new growth with cavity formation in the Spinal Cord. Brain, Pt. LXXV., 1896.

¹⁰Babes et Manicatide. Recherches sur la Syringomyelie. Archives de Sci. Med. de Bucharest, May, 1896.

¹¹J. Prus. Die Morvan'sche Krankheit, ihr Verhältniss zur Syringomyelie und Lepa. Archiv. für Psych., XXVI., 771, Nov., 1895.

Periscope.

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LUMBAR PUNCTURE.

Caillé (Archives of Pediatrics, Aug., 1896) reports eighteen cases in addition to the three which he has already reported. No difficulty was encountered in puncturing, no anæsthetic was employed, and in only two cases did a rise in temperature follow the operation. In most of the cases the pressure of the fluid was such as to force out the piston of the syringe.

The author considers the operation easy to perform and of very considerable diagnostic value. He has made an attempt to add to it a therapeutic measure, as shown by the following paragraphs.

"It will naturally occur to any one working in this line that a liquid may be just as readily injected into the spinal canal as it is removed therefrom, particularly after the pressure of the fluid has been diminished.

"This I have done in two cases with a view of favorably influencing the course of an otherwise incurable tubercular meningitis and in hopes of gaining somewhat similar results as we obtain in the local treatment of tubercular peritonitis.

"The two cases prove to me that a more thorough washing of the sub-arachnoid space is necessary in order to make an impression upon a case of tubercular meningitis. At the next opportunity which presents itself I propose to lay bare the dura by removing a button of bone with the trephine and irrigate the sub-arachnoid space from a lumbar puncture upward through an opening in the dura. Irrigation by the shorter route through the lateral ventricles will probably not reach the convexity and will be inadequate.

Wentworth (Archives of Pediatrics, Aug., 1896) has done some valuable experimental work on healthy children and on those affected with some disease not involving the nervous system, from which he concludes:—

1. "The normal cerebro-spinal fluid contains neither cells nor fibrin, and is perfectly clear.

2. "In cases of meningitis (?) the cerebro-spinal fluid is invariably cloudy when withdrawn. The degree of cloudiness is to some extent proportionate to the amount and character of the exudation in the meninges.

3. "The cloudiness is caused by cells. The character of the cells differs with the variety of the meningitis. After withdrawal more or less fibrin is formed in the fluid. The presence of these cells and fibrin is pathognomonic of inflammation in the meninges.

4. "The cloudiness is oftentimes so slight that close observation is necessary to detect it.

5. "The operation is not difficult to perform on infants and children. It is not dangerous if strict cleanliness is observed.

6. "A differential diagnosis between the various kinds of meningitis can be made by microscopic examination of the sediment, by cultures taken from the fluid and by inoculation experiments."

7. "Inoculation experiments afford the surest means of determining tubercular meningitis. It is of value to distinguish between the varieties of meningitis in order to determine if tubercular meningitis is recovered from.

8. "In the normal fluid a trace of albumin is usually present, about $\frac{1}{10}$ of one per cent. or less by quantitative analysis. In meningitis the amount of albumin is increased and has varied from $\frac{1}{10}$ to $\frac{1}{5}$ of one per cent.

9. "In one case a diagnosis of general infection with the staphylococcus pyogenes aureus was made from cultures taken from the cerebro-spinal fluid."

He has performed the operation forty-five times, and has never seen any ill effects. Regarding the operation itself, the following remarks may be quoted:—

"An antitoxin needle is preferable to an ordinary hypodermic needle; it is less liable to break and has a larger lumen, besides being somewhat longer. The one used on children over three years of age was four and one-half centimetres long, with a diameter of one and one-half millimetres. For infants under three, a needle four centimetres long, with a diameter of one millimetre, was used.

"A syringe is never necessary, but it is well to have a sterile wire to pass through the needle in situ, in case the fluid does not run well."

He is in accord with some other observers in concluding that "the degree of force with which the fluid is expelled through the needle has little diagnostic value as indicating an increased amount of fluid. I have seen the fluid spurt in a fine stream in several cases in which there were no brain lesions; and, on the other hand, it has dropped from the needle in most of the cases of meningitis and in one case of hydrocephalus. In this latter case when it was punctured a second time the fluid spurted at first."

Notwithstanding the positive assertion that in meningitis the fluid is always cloudy, he says:—

"It is possible in the beginning of a tubercular meningitis to obtain a perfectly clear fluid. This was exemplified in one case in which two subsequent punctures showed the fluid to be slightly cloudy and to contain small round cells and fibrin. At the autopsy of this case only a few miliary tubercles were found at the vortex of the brain and but a little inflammatory exudation, also at the vortex."

So far as we know, the finding alluded to in the following paragraph has been mentioned by no other author:—

"In a certain number of cases numerous white particles were present in the fluid when it was withdrawn. They were neither cells nor fibrin, and showed a tendency to dissolve after several hours. Solutions of corrosive sublimate and alcohol were added to normal fluid without causing the particles to appear. The skin was moistened and scraped and the scrapings examined also with a negative result. These particles appeared in the fluid withdrawn from cases in which there was no meningitis. They should not be confounded with the cloudiness due to cells. The latter is very finely divided and gives a general haziness to the fluid. The particles may occur in cases of meningitis, but they do not interfere with the detection of the general cloudiness."

Jennings (*Archives of Pediatrics*, August, 1896) relates a case of supposed tubercular meningitis in a child of six years. At the time of the

puncture she was comatose, with dilated, uneven and sluggish pupils, gummy conjunctival secretion, general tremor,—the usual phenomena of the stage of effusion. A large antitoxin needle was used, and 24 c. c. of fluid were withdrawn, but the fluid flowed freely only when the needle was depressed. Too much force was accidentally used in depressing the needle, and it broke deeply in the tissues of the back. A deep incision and thorough search failed to reveal the fragment. A few hours after the puncture the temperature rose to 104.8 and the pulse to 136. Up to that time the temperature had not risen above 101 or the pulse above 100. On the following day the puncture was repeated and eight drachms of fluid withdrawn. After the second puncture there was a partial return of consciousness and temporary relief of the pressure phenomena. She died, however, two days later. No autopsy. The fluid withdrawn was clear, depositing a few flocculi of fibrin after standing. Pneumococci were present in large numbers.

In the discussion of the foregoing papers Dr Fruitnight said that he had seen the operation done repeatedly without bad results. Dr Blackader had resorted to the procedure two or three times, and in one case there seemed to be a very decided improvement in the symptoms for some time. Dr. Holt said that he had employed the puncture three times and had removed as much as three ounces of fluid, but had observed no change in the symptoms. He thought the operation was not likely to prove of great value in diagnosis. Dr. Wentworth remarked that the inoculation method was the only sure way to control the diagnosis and to learn positively whether tubercular meningitis was ever recovered from. So far, in cases which have recovered, the diagnosis has depended upon cover glass preparations, where a mistake in diagnosis was possible.

Babcock (State Hospital Bulletin, N. Y., July, 1896) reports on nineteen cases in asylum practice in which puncture was done. The operation was performed in twelve cases of general paralysis, in two cases of simple melancholia with pressure symptoms (intense headache, stupor, photophobia), and one case each of locomotor ataxia, stuporous melancholia, organic dementia and status epilepticus.

The author ordinarily used only local anæsthesia (cocaine injections), and agrees with others that the operation is not very difficult, nor has he seen unpleasant consequences, except intense transitory headache. Queerly enough, he seems to think that the needle is to be inserted alongside of the cord, whereas, as is well known, the cord is above the site of the puncture,—even if this be made, as was sometimes done by Babcock, between the first and second lumbar vertebra. This is, however, unnecessarily close to the cord, and we would counsel a lower site. The therapeutic results of the operation cannot be considered very good. Some cases were entirely unaffected by it. Some showed a slight improvement for a few hours, and in only two (one of general paralysis and one of subacute mania with locomotor ataxia) was there decided improvement lasting for several weeks (to the time of the report). In the second of these it is a question how much the operation had to do with the improvement, as this did not begin until a week after the puncture, ample time for reaccumulation of the amount of fluid (95 c.c.) removed. We would conclude from the report that lumbar puncture in the insane possibly constitutes a legitimate field for experiment, but that at present it promises to add very little to the therapy of psychiatry. PATRICK (Chicago).

OPHTHALMOPLÉGIC MIGRAINE (PERIODICAL OCULOMOTOR PARALYSIS)

Dr. d'Alchè (*Jour. de Med. et de Chir. prat.*, July 25, 1896) was able up to the present time to collect the reports of twenty-five cases in the literature bearing upon this disorder. First described in Germany, ophthalmoplegic migraine was then studied and published in France by Parinaud and Marie, afterwards by Charcot and Ballet. According to

Charcot the malady is not an entity, but a symptomatic complex, characterized by the presence of hemicrania with concurrent total paralysis of one of the oculomotor nerves. The one sided head-ache is quite characteristic, and emphasized by the various observers. The intensity of the pain is seldom very great, but deeply seated, continuous, with morning or evening exacerbations, and extending from the occiput to the orbit. The ocular paralysis sets in after the development of hemicrania; it is invariably unilateral, and nearly always a complete palsy. Another characteristic feature, observed by all, is the tendency and periodicity of relapses, as for instance, once or twice yearly, every three months, etc. In one case (Moebius) every August for three consecutive years, in another, (Thomsen) in May and October. After a certain length of time, the disease undergoes a change, the symptoms becoming more permanent and continuous. 1. Periodical stage; 2. Permanent stage. A typical attack of *migraine ophthalmoplegique* presents, during the *painful period*, the same features as ordinary migraine (nausea, vomiting, photophobia and hemicrania), at times with oedema of the eyelids, and salivation. The duration is quite variable and sooner or later exacerbations intervene. The onset is generally sudden without any definite cause.

The *paralytic period* sets in generally several hours or days, or, rarely, weeks after the painful and gastric symptoms. The upper eyelid begins to droop, the bulb deviates externally and the whole distribution of one of the third nerves is impaired (paralysis of the iris, the muscles of accommodation as well as the external eye muscles). The development of these phenomena is rapid without being sudden as in cerebral hemorrhage. The painful symptoms (migraine) usually continue during the paralytic stage for some time, but are less marked or intense, which fact is in opposition to Charcot, who considered the onset of the latter period as indicative of cessations of the former. The paralytic troubles may last for hours or months, disappearing as they set in, rapidly, the patient feeling as well as ever after the attack. New attacks are always to be anticipated, which invariably have the same characteristics as the preceding one.

As the malady grows older it takes a chronic character (Thomsen's case lasted for 29 years), the paralytic attacks becoming rarer but more tenacious, and in the intervals between, the symptoms persistent with periods of exacerbations, which gives the malady the character of a continuous disorder with periodical exacerbations. The tendency to become aggravated is far greater than to recover, although there may be a possibility of a spontaneous cure. The ætiology and pathology are quite obscure, although the subject of considerable speculation. The treatment consists in the prolonged administration of bromides (Charcot) with potassium iodide for circulatory disturbances, and electricity. The last especially when the paralysis of the eye muscles is permanent.

MACALESTER.

A CASE OF TABES DORSALIS WITH BULBAR SYMPTOMS.

Deutsche Zeitschrift für Nervenheilkunde. Vol. 9, Nos. 1 and 2, 1896. By H. Grabower, M. D.

In a case of tabes the following conditions were found. Degeneration of the left recurrent nerve and of the extrabulbar portion of the roots of both tenth nerves, perfect integrity of the roots of both eleventh nerves, degeneration of the roots of both ninth nerves, integrity of the nuclei and intrabulbar portion of the roots of the tenth and eleventh nerves, moderate degeneration of both spinal roots of the fifth nerves. The nuclei of the third, fourth and sixth nerves were atrophied.

The author is inclined to accept the theory that in certain cases the tabetic process begins in the peripheral sensory nerves, and ascends to

the posterior roots and posterior columns. The spinal ganglia may be altered functionally without presenting organic changes.

On account of the disease of the sensory nerves, the cells of the spinal ganglia are deprived of the normal impulses from the periphery, and undergo the atrophy of inactivity, which however may not be demonstrable (Marinesco)

To explain the involvement of the motor nerves, as of the vagus in this case when its nucleus was intact, it is necessary to suppose that the morbid process is transmitted by means of the sensory roots to the cells of the anterior horns in the cord, and within the oblongata to the motor nuclei. These are altered functionally, but not organically. In this case the sensory descending roots of the ninth and fifth nerves were degenerated.

The case is of great value in regard to the motor innervation of the larynx. It has been a disputed question as to whether this is a function of the vagus or of the accessorius. In 1890 Grabower from experiments reached the conclusion that the accessorius has absolutely nothing to do with the innervation of the larynx, and that the vagus is its only motor nerve. Similar statements were made by Grossman.

Grabower has shown by means of serial sections that the nucleus of the accessorius passes into that of the hypoglossus, that a cerebral accessorius nucleus does not exist, that from where the nucleus of the accessorius terminates to where the motor nucleus of the vagus begins there is a space of more than nine millimetres, and that there is not the slightest connection between the vagus and accessorius nuclei.

There are sixteen cases known to Grabower in which paralysis of the larynx was observed in connection with paralysis of the trapezius. Seven of these are undoubtedly of peripheral origin and useless therefore for the question at issue, for a process which involves the accessorius after its exit from the jugular foramen is apt to involve the vagus also. In none of the nine remaining cases has an autopsy been reported.

In Grabower's case of paralysis of the laryngeal muscles the extrabulbar part on of the roots of the tenth nerves were found almost completely atrophied, while the roots of the eleventh nerves were perfectly intact. This according to the author, proves that the accessorius has nothing to do with the innervation of the larynx.

Oppenheim in speaking of this case, says that Grabower has demonstrated that the innervation of the larynx is through the vagus and not through the accessorius. He believes the tabetic virus affected the vagi roots, and not the nuclei in a manner similar to the action of lead on the radial nerve, although he acknowledges the difficulty in deciding this point.

Pharyngeal crises, which were observed in this case, were probably due to the degeneration of the ninth nerves.

The degeneration of the spinal roots of the fifth nerves explains the analgesia of the face and the loss of the teeth observed in this case.

SPILLER.

ARTHROPATHY AS THE EARLIEST SYMPTOM OF TABES DORSALIS.

F. DE GRANDMAISON (*Médecine Moderne*, Sept. 30, 1896,) describes two cases in which the earliest conspicuous symptoms of ataxia were the appearance of articular enlargements at the knee. One patient was a man of fifty years, the other a woman of thirty-five. The diagnosis of ataxia was made in both cases on two cardinal symptoms, the total absence of knee-jerk and the presence of the Argyll Robertson phenomenon. Neither patient had any disorder of station, neither had suffered with any crises of pain, so that both may be considered to have had remarkably early articular manifestations, although it will be remembered that Charcot insisted upon the frequency with which arthropathies appeared in the pre-ataxic stage.

MITCHELL

OXALATE OF CERIUM FOR GASTRIC CRISES.

Dr. Ostankow, of St. Petersburg, is quoted in the *Sem. Médicale* June 18, 1896, as having found oxalate of cerium, hitherto only employed for troublesome vomiting during pregnancy very useful in the troublesome gastritis of ataxic patients. He gives it in moderate doses three or four times a day, and finds it lessens the frequency of the vomiting and relieves the gastric pains and the thirst which is so intense during the crisis.

MITCHELL.

TREATMENT OF THE GASTRIC CRISES IN TABES.

Revista de Ciencias Médicas de Barcelona, May 25, 1896.

In the clinic of nervous diseases of Prof. Becntere, the oxalate of cerium is employed in cases of locomotor ataxia with gastric crises, in doses of 0.05 to 0.15 four times daily. The author has noted especially its effect in allaying nausea and vomiting. The attacks of vomiting were reduced in some cases from 200 in 24 hours, to 6 or 8; on the third day or even less. The gravity of the attack was also lessened, that is the pain referred to the stomach, the nausea, and the great thirst. Sometimes the psychological state of the patient is quieted permitting him to sleep soundly, and to urinate without catheterization.

W. C. K.

THYROID FEEDING IN THE INSANE.

Chas. G. Hill, (*Maryland Med. Jour.*, Sept., 1896) reports the results of thyroid treatment in forty insane patients. The character of the cases is not precisely given, the author simply stating that the experiments were made on a wide range of subjects, from the acute forms of mania and melancholia to the long abandoned dement. but the bulk of the cases were those of dementia under 50 years of age and of not too long standing.

The summary of results given is: unimproved, 8; improved, 12; greatly improved, 14; cured, 5; died, 1. The following cases presumably, the most striking are the only ones detailed:

"L," about thirty-five, a dement of five years' standing, fat, passive sluggish and absolutely silent, as his voice had not been heard during all this time except on one memorable occasion when he cried out from pain. In forty-eight hours after commencing the thyroid in doses of ten grains three times daily, he was not only conversing freely and intelligently, but swearing like the "army in Flanders" at any one who molested him. In spite of persistent treatment for some months he relapsed gradually into his former condition, except that he will reply to questions, but never voluntarily enters into a conversation.

"F.," a profound melancholic of one year's standing, silent and immovable as the sphynx, and morose to an extreme degree, in two days was picking husks for a mattress, but so hilarious and talkative as to appear hysterical. He has never relapsed into his melancholia, has been an industrious and exemplary patient, and will probably recover in the end.

"I.," another melancholic, who was always improved by the use of opium, but relapsed as soon as it was withdrawn, even though done in a most gradual manner, on suddenly withdrawing the opium and substituting the thyroid, recovered and was sent home within a month, and has remained well.

"G and P," dement of seven and fifteen years' standing, respectively, and between forty and fifty years' of age, were made so sick by its use (which caused vomiting, profuse perspiration and great heart depression) that its administration had to be discontinued and there was no perceptible improvement in either case.

"M. and R.," young men in their twenties, both suffering from

acute mania, characterized by violent outbursts and exacerbations, one of six and the other of eighteen months' standing, in whom all treatment had so far failed to afford relief, were both cured and have returned to their homes.

F., a strong, well-built man, suffering from acute mania of a violent type, who talked, swore or sang incessantly, broke up everything in his reach and could not be controlled by either mechanical or chemical restraint, or both combined. Bromides, chloral, hyoscyne, cannabis indica or morphia, even when pushed to the verge of danger failed to exert any influence over him, but in twenty-four hours after beginning the use of the thyroid, he was quite and docile and has still remained so. He has interested himself in work on the farm and would have been discharged but for some delusion that seems difficult to eradicate.

"M," a case of chronic mania, became so violently excited that the treatment was discontinued—result negative.

A very interesting case is that of Mrs. G, a young married woman, aged about thirty. Seven years ago she was admitted to Mt Hope as a case of acute mania, and in spite of all treatment, relapsed into dementia within twelve months. Two years afterwards, on being told that her case was hopeless, her husband removed her to another institution as a matter of economy. Three years later, the finances of the family having improved, she was returned to Mt Hope, the same silent, helpless dement as when she left, and up to the beginning of the thyroid treatment, two years afterwards, remained in this condition. She was as helpless as an infant, dirty in her habits, had to be dressed and undressed and taken to and from the dining-room. The effect of the thyroid treatment with her was magical. She was talking the next day, dressed and undressed herself the second day, cleaned up her room the next morning and asked for some work to keep the time from hanging heavily in her hands. Though the treatment has been discontinued, she continues to improve slowly.

PATRICK (Chicago).

CRETINISM AND ITS TREATMENT BY THYROID GLAND.

Parker (*Brit. Med. Jour.*, Sept. 12, 1896,) after an extensive experience in the thyroid treatment of cretinism, notes the following results:

"1. A great and rapid diminution of bulk, due to absorption of myxœdematous deposits, seen especially in the collapse of the protruding abdomen, in the spontaneous reduction of umbilical herniæ, in the recession of the previously swollen tongue behind the teeth, in the disappearance of baggy swellings under the chin, above the collar bones, outside the nipples, and elsewhere, in the thinning of the lips, and in the disappearance of the dropsy-like puffiness of the face, limbs, and other parts of the body.

"2. A great and rapid increase in physical development, shown especially by a rapid growth of several inches in height, even in cretins of from 20 to 30 years of age, whose stature had been nearly or quite stationary for many years previously, also by the replacement of the coarse sparse hair by a more abundant and finer growth, by the eruption in quick succession of teeth which had been long overdue, as in cases where the milk teeth still persisted at 20 years of age, and by a substantial increase of body weight, after the initial loss from absorption of myxœdematous deposits.

"3. A striking diminution of several hideous deformities, especially of the lordosis in the lumbar spine, of the bulky head, of the ugly sinking of the bridge of the nose, and sometimes of the rickety curvature of the legs. Many of the pictures, however, showed little or no improvement in the deformity of the legs, owing to the softening of bones produced by thyroid extract, and to the fact that the majority of cretins were allowed an undue use of their legs during treatment.

"4. A rapid and very striking increase of intelligence occurred, as was well seen by comparing the dull, stupid, heavy, listless, often idiotic countenance before treatment with the bright, cheerful, pleasing expression which soon took its place."

Telford-Smith in the discussion called attention to the similarity of idiots of the Mongol type to cretins, both in physical aspects and mental characteristics.

"In idiots of this type we get the stunted growth, the dull, heavy expression, with open mouth and thick lips, the slow deliberate movement, and hoarse, guttural, and monosyllabic speech, the mental apathy, and lack of spontaneity, the sluggish circulation and sensitiveness to cold. A thickened condition of subcutaneous tissue is often found, with dulled cutaneous sensibility. The skin is coarse and dry, the hair short and thin. First and second dentition are delayed. As far as palpitation enables one to judge, the thyroid gland is subnormal in size. The temperature is always subnormal, ranging between 96.5° and 97.5°."

After two years' trial of thyroid in the Mongol idiot the author summarizes his experience as follows:

Improvement of a physical and mental kind takes place. This varies inversely as the age of the patient. The improvement is not nearly so marked nor so rapid as in the case of the cretin. The temperature reacts, but only slightly; it cannot be kept at normal. There is some improvement in the condition of the skin; it desquamates but does not become normally smooth. Tarsal ophthalmia (marginal blepharitis) which in these cases is almost chronic, improves considerably, and in some cases is cured. Growth improves, but does not react in anything like the rapid manner seen in cretinism. The mental condition of the child improves; the apathy is less pronounced; the patient becomes more active and spontaneous in his movements; he plays more like other children, and joins in the simple amusements or employments going on around him; he even works voluntarily. His speech is less thick and guttural, and he talks and chatters to a more normal extent. His mental reflexes all seem more active, and there is a decided advance on the patient's previous rather vegetable existence.

In other cases of idiocy in children of eight to nine years he noted some improvement but it was not striking.

A practical point in the treatment is that it sometimes causes such rapid growth of the skeleton as to make the bones soft, so that if the child be on its feet much during the treatment, the leg bones are apt to become bent. This necessitates treating some cases in bed.

Thomson found the rapidity of growth to bear an inverse ratio to the age of the patient and that children grew much more the first year of treatment than during any succeeding year. He also had noticed marked bending of the legs and great increase of already existing lateral spinal curvature. The character of the hair changed in children, but not in adults. Marked mental improvement occurred only in children, not in adolescents or adults.

Victor Horsley showed a photograph of a well-marked specimen of intrauterine cretinism. The child was still-born.

Fletcher Beech presented an exhaustive analysis of 116 cases with reference to etiology. It is impossible to abstract the paper, but it may be said that no salient etiological factor was found. Consanguinity, intemperance, phthisis, inheritance of mental disease, are perhaps the most important. Parents often ascribe the disease to fright of the mother during pregnancy, but this cause is problematical.

Bury had seen no results of thyroid treatment in two cases of Mongol idiots. He related the interesting case of a child who at the age of one year ceased to "get on" and showed signs of incipient cretinism. One-fourth tabloid daily "speedily picked him up," and after six months, treatment was discontinued without relapse. Although there must be

some doubt as to the diagnosis in this case, it is of interest as tending to show that, if a case of cretinism be taken sufficiently early, treatment need not be kept up indefinitely. Up to the present time experience has shown that when thyroid treatment is stopped, the child retrogrades.

W. H. George, in the same journal, reports a case of cretinism treated by thyroid. The patient, a girl of 12, 2 ft 9 in in height, had not grown in six years and showed all the signs of cretinism. She was given about a 5 grain tabloid daily. After a year and one month of treatment the child had grown 2½ inches, had cut a permanent molar and could talk fairly well. She was speechless before the beginning of treatment.

PATRICK, (Chicago).

THE PATHOLOGY OF EXOPHTHALMIC GOITRE.

Murray, in opening a discussion on the pathology of Graves' disease (*Brit. Med. Jour.*, Oct. 3, 1896) concluded that the cause of the disease was to be found in the thyroid gland, and he knew of no case in which this was found normal after death. He first compared the histology of the normal thyroid with thyroid tissue in a state of compensatory hypertrophy. The latter showed the formation of new alveoli and great increase of epithelial elements; folding of alveolar walls which increased the secreting surface; change of the epithelial cells from cubical to columnar form. These changes are entirely comparable to those found in Graves' disease, and he says:

"In conclusion I would maintain that in exophthalmic goitre there is an excessive formation and absorption of thyroid secretion, which may or may not be normal in character, and that the symptoms of the disease are due to the presence of this excess of secretion in the blood, and to its action upon the tissues and especially upon the nerve centres in the medulla.

The practical conclusion from this is that we should endeavor to improve our methods of treating the diseased thyroid gland. We want to be able to induce a moderate degree of fibrosis, and so imitate the natural process of recovery. Removal of a portion of the gland, though most successful in some cases, has proved to be a dangerous operation in others. Possibly injections of iodine, electrolysis, or some similar means of starting a limited fibrosis, may help us to attain the object we have in view without danger."

Edmunds agreed that changes of the thyroid in exophthalmic goitre are practically identical with those found in a part of the gland which has undergone hypertrophy in consequence of removal of a larger part of the gland, but unlike Murray he concludes from this that in Graves' disease too, the changes are simply those of compensatory hypertrophy, in other words, the change in the thyroid is not the primary lesion. He thought, too, that the disease cannot be due to hypersecretion of normal thyroid juice, because if this were the case there would be no occasion for a compensatory hypertrophy. Although it is easy to draw a contrast between myxœdema and exophthalmic goitre and to conclude that because the first disease is due to a decrease of thyroid secretion the second must be due to an excess, there are objections to the theory.

"First, although the contrast between the symptoms of Graves's disease and myxœdema is well marked in the chronic forms, in the acute form (as seen in dogs) it is not at all marked; indeed the two affections resemble one another. In both these are well-marked tremors and occasional attacks of dyspnoea. Secondly, the theory of excessive secretion will not explain the exophthalmos; the injection subcutaneously of thyroid extract in large quantities into monkeys does not produce exophthalmos in these animals, although the condition can be readily enough produced in them by the injection of cocaine. Thirdly, the theory will not explain how in many recorded cases the exophthalmos is on one

side, or mainly on one side and the enlargement of the thyroid marked on that side. Fourthly, the not uncommon occurrence of myxoedema following Graves's disease seems to support the secretion view; but the theory would require an interval of good health in the transitional stage. One finds mention of cases in which the two diseases appear to co-exist. Finally, against the secretion theory is the effect of thyroid feeding; different results have been obtained and the majority of observations seem to show that the treatment does no good, but it does not seem to do harm with the certainty that might be expected were the symptoms due to an excess of thyroid secretion. The primary lesion is a derangement of the metabolism of the body."

Robert Hutchison had concluded from experiments and clinical observations that the colloid matter of the thyroid was its only active ingredient, Baumann's thyroïdin (thyroïodin?) being an artificial product and representing only part of it. He had entirely failed to find an alkalioid in the thyroid. To prove the thyroid origin of Graves's disease it must be shown that there is overproduction of colloid or that there is qualitative change in the secretion. As to the first, pathologists are agreed that no increase of colloid is observed in glands removed from patients with exophthalmic goitre and there is no evidence tending to show that the secretion is removed from the sacs more rapidly in that disease than in the normal individual. As to the second condition, the only practical way to test it is to try the physiological effects of the different ingredients of thyroids taken from cases of exophthalmic goitre. This he had done. By appropriate treatment the different ingredients were separated from a thyroid gland weighing 85 grammes, removed by operation from a patient with typical exophthalmic goitre. These were administered to two young women who were receiving thyroid treatment for a skin affection and obesity, respectively. The proteid-free extract was found to be inert. The colloid matter caused loss of weight, slight rise of temperature and some acceleration of the pulse. These symptoms were not more marked than had been produced by equivalent doses of sheep's thyroid to the same individuals. No palpitation was complained of in either case, nor was there the least degree of exophthalmos produced, or any appreciable alteration of the thyroid. To a dog, from which the thyroid had previously been removed, larger doses of the colloid (8 grammes of fresh gland daily, or about as much as 1½ thyroid of an average sheep) were given. The temperature of both showed a slight rise but no other symptom was produced. As far as these observations went they were directly opposed to the view that the thyroid in exophthalmic goitre produced a peculiar secretion.

"In conclusion, Dr. Hutchison was inclined to regard the disease, exophthalmic goitre, as consisting essentially in a specific change in tissue metabolism, probably due to a functional alteration in the central nervous system, the relationship of the enlargement of the thyroid to these changes being as yet undetermined, but not to be regarded as their primary cause. The beneficial effects of surgical operation in exophthalmic goitre were not necessarily opposed to this view. One might tumble downstairs both from the action of gravity and also because he was propelled by a kick from behind. Withdraw the propulsion of the kick and gravity itself would not start the descent. So it might be in exophthalmic goitre. Remove that tendency to acceleration of tissue metabolism which the normal secretion of the thyroid had been proved to produce, and even a functional loss of control on the part of the nervous system might be unable to start that train of metabolic changes which lead to the symptoms of exophthalmic goitre."

PATRICK (Chicago.)

American Psychiatry.

UNDER THE DIRECTION OF

R. M. PHELPS, A. M., M. D.,

Rochester, Minn.

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PROCEEDINGS OF THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION. VOL. 2. ANNUAL MEETING JUNE 11-13, 1895.

This is a most compact volume of 258 pages. Volume 1 incorporated the proceedings of 1894, upon the fiftieth anniversary of the "Superintendent's Association." For several years previous to this meeting, under the leading effort of Dr. Cowles, Dr. Hurd and others, the association had been growing out of its old organization as "Superintendents," and adopting itself to its new designation, "Medico-Psychological," under which limitations and conditions it is now working. It was at this meeting that Dr. Mitchell delivered the caustic address which the members claimed probed not only some real wounds, but in a disorganizing way far beyond them into structures ordinarily healthy.

The special articles do not need much of detailed comment, having been before the public so recently. A running commentary only will be attempted.

Dr. Cowles' article on custodial and medical management is the most important. He sturdily maintains as appropriate the medical management of hospitals, the superintendent being a "co-ordinating" power, bringing all departments to serve medical aims. Farming, build-

ing, carpentry, gardening and other businesses he presumably includes, though they are not specifically mentioned. To make this scheme thoroughly medical, however, he would have assistants of ability and experience who should share in the government of the hospital, so as to fit them to become superintendents. He would have a hospital medical society, which would co-ordinate and stimulate the medical work. He shows quite clearly the injustice of some current criticisms or comparisons, but this we cannot here take space to detail.

The article of Dr. Wise on medical work in hospitals for insane will be more practical to many. He outlines a thoroughly medical work, ignoring the large business interests; the superintendent being the director; daily staff meetings and medical discussions keeping all in touch; routine "rounds" superceded by clinical studies; conventional "case books" set aside for medical nursing records; clerks (not physicians) to do all copy writing; a well developed training school, and nurses making steady and constant record of observations on all new cases, all of which records become permanent. By such accounts as these it will be noted that the observations and records of cases in our best hospitals for insane are as detailed and laborious as in any other hospital service.

In the class of clinical medicine, we have papers on thyroid feeding by Dr. C. K. Clarke and Dr. Edward Brush, each giving experience with cases and reporting mildly hopeful results.

Dr. Waughop presents a case of double Hemianopsia, and Dr. G. H. Rohe again maintains his belief "in the causative relation of pelvic diseases and insanity in women," and reports a certain proportion of recoveries after the surgical removal of the uterine appendages. (The effect of non-surgical treatment or minor surgical operations is not discussed.) In thirty cases so operated on, ten are reported cured mentally, four decidedly improved, thirteen unimproved, three died. One lack in this report is a failure to give a detailed list of physical conditions found in these operations. The "significance of motor disturbances in insanity," by Dr. A. B. Richardson, contains a cursory review of such disturbances.

Of papers partially custodial in character, we find articles on "Asylum Dietaries," by Dr. J. D. Munson and Dr. Charles W. Pilgrim. Dr. Munson records a laborious and painstaking attempt to study the food supply scientifically, in its quantity, quality and constituent elements as well as in its preparation. A *chef* is engaged as in charge of all cooking, the food is served in more tempting forms and dining rooms are better furnished. The Kankakee Hospital is also working along these lines. This is to our own minds an argument that the growth of "medical" spirit brings better "custodial" condition than the custodial by itself.

A report of some interesting pathological findings was made by Dr. Adolph Meyer, and Dr. H. A. Berkeley presented some "studies on the lesions produced by the action of certain poisons on the cortical nerve cell," this being a part of his recent work so widely reviewed as not to need further comment.

Daniel Clark (Toronto) presents nine medico-legal cases prominent in Canadian courts. These will be found very instructive, though without the study of physical stigmata, which should not be overlooked. The Riel, the head of the rebellion of the Northwest Territories is a more case of interesting and varied one than that of Guiteau. It emphasizes the fact that a certain number of ill-balanced and insane persons are always abroad in the communities surrounding us,—and that insanity is not an entity, but more truly a symptomatic abnormality.

Dr. Blumer and Dr. H. A. Bannister, appointed to delineate the influence of alcohol drinking upon insanity, report that alcoholic excesses directly cause at least 10 or 12 per cent. of insanity; that there is no reason to believe that moderate drinking is conducive to health, but

that there is a presumption to the contrary, hereditary and unstable cases being especially referred to.

There were not presented quite so many of clinical studies as at the preceding meeting, but Pathological studies were more prominent than usual. It seems pertinent to testify in conclusion that a steady and rapid growth in hospital work is at present in progress, and will undoubtedly be manifested the literature embodying the results obtained. This meeting was in Denver, and on account of the distance many prominent members were absent.

PHELPS.

STATE HOSPITAL BULLETIN.

Volume I, No. 1 of the promised State Hospital Bulletin, (N. Y.), reaches us at the end of February. It presents 140 pages of essays arising from a study by various medical officers, each of his own clinical material. The actual material presented is almost secondary to the possibilities hinted at in the general scheme. For example, if the central editorial committee has influence or authority to divide up the experimental and research work among the different hospitals and medical officers, it can develop along many lines, by such division of labor. For example, assigning thyroid feeding to one hospital, urinalysis to another, etc. The practicability of the complete development of this scheme is more assured than its desirability. It would perhaps not wholly suit independent thought and tastes.

Editorially it is promised to develop the pathological work during the coming year, in a manner spoken of by them as "ideal." A central bureau known as the Pathological Institute will under Doctor Van Gieson direct the work of the local hospital laboratories, seemingly assigning them research work something as above outlined. It is then not work in a central laboratory, *under the direction of* a central laboratory. Details are not given, but the outlook is good. Considerable time is needed to get results from this system.

For its further aims it is stated that "it may be considered in the light of a "psychiatrist at the breakfast table" and as it is not intended to compete with the formal Journals, so it should not be compared with them in the character of its contents. Articles which are not suited for the ordinary journals will find place in the Bulletin. Negative results will be recorded,-- results in therapeutics and physical treatment will be freely used."

Notice of the essays comprising this number is precluded here by the lateness of its arrival. It is published by the State, by the "Utica Hospital Print," seemingly as was the Journal of Insanity. No advertisements are taken and the subscription price is \$2.00. As a whole the plan promises well, not only by publishing present results but by setting each hospital at work on new or renewed lines.

PHELPS.

A NEW TEXT BOOK FOR TRAINING SCHOOLS FOR NURSES,

By Dr. P. M. Wise, is just at hand. It accepts seemingly the same reasoning adopted by the writer two years ago; namely, that a book to study by and follow was better for students of this grade and with this amount of time, than such insufficient "notes" as they could make; that the nursing of mental and bodily diseases are best woven together into one whole; that a "book" secures a consistent and known line of study, even if the lecturers be several or changing.

Some differential points between the two books might be mentioned. This later book leaves out of consideration special drugs, perhaps wisely so. It has an index, glossary and some tables, as very valuable additions. Peculiarly enough, while giving a space to obstetrics, (we think rightly,) it omits all consideration of gynecological work, a subject of almost daily practical use in hospital service. It also omits con-

sideration of electrical applications; also of mechanical restraint, a question that must be met by every nurse, and intelligently rather than ignorantly. It gives more of detail to the treatment of insanity and less to general nursing. It gives nothing about the "examination of urine" or "control of fire," nor has it any sketch of the development of Insane Asylum work or methods and of the comparative value of the different schools. He adds, however, more of detail on the subjects of "Sleep" and "Sleeplessness," "Anæsthetization," "Micro-organisms," convulsions, and anatomy of brain, nerves and spinal cord. Nothing is said about menstruation and its disorders.

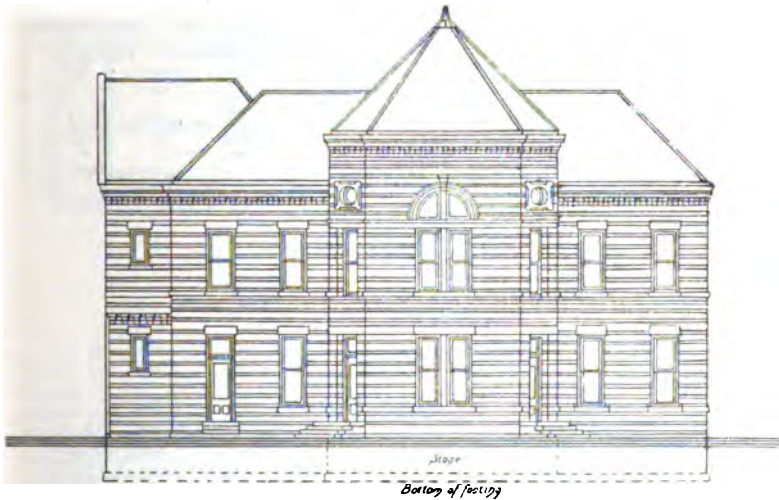
The book is bound in two volumes, one for each year of study, and its value is very considerably enhanced by illustrations. The book will doubtless meet with great favor, indeed (according to the advertisement), being adopted by the New York Hospitals before it was issued. The name of Dr. Wise, whose hospital ranks among the very first in the country, is sufficient assurance of acceptable and reliable work.

PHELPS.

THE NEW PATHOLOGICAL INSTITUTE OF THE CENTRAL INDIANA HOSPITAL FOR THE INSANE.

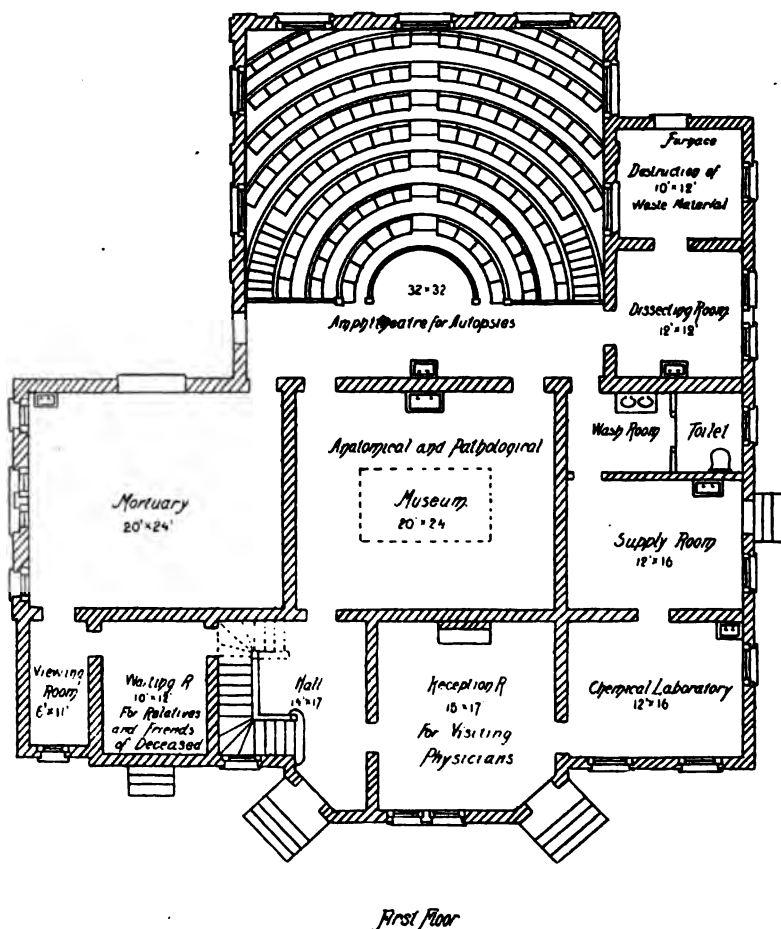
An event of considerable importance to the local medical profession and of interest to neurologists and alienists generally, will be consummated within the next few weeks, when the new Pathological Department of the Central Hospital for the Insane is dedicated to original work and research. With the completion of this building and its thorough outfit for all kinds of chemical, bacteriological and pathological investigation, Indiana has taken a decided step in advance, and placed herself upon a high place, one equal to that held by the best Hospitals and State institutions of this country and Europe. The recent official report of the Central Indiana Hospital for the Insane dwells especially upon this new department, offering, as it does, opportunity for the study and careful systematic investigation of mental and nervous diseases.

The structure of the new building in itself is admirable. Built substantially of red brick ornamented with stone, the external appearance of the building is beautiful in its garden surroundings. The interior is a model in its arrangements and finish. There are reception rooms for physicians and visitors upon the ground floor, and also a neatly furnished mortuary, where relatives and friends of the dead may view the remains without any shock to their sentiments, a point of importance in lessening the wide-spread opposition to post-mortems existing in this country. All the other rooms of the building are devoted to medical research. Upon the lower floor are the chemical laboratories, the dissecting and preserving room for the examination of microscopic specimens. In addition there are here situated the reagent room, the museum, and the clinical amphitheatre, in which autopsies will also be held before members of the profession and seniors of the medical schools. The amphitheatre is large, excellently ventilated, and lighted by drop and side electric lamps. Several thirty-two candle hand-lamps add materially to the possibilities for illuminating the body cavities. Every seat is so arranged that the occupant has an uninterrupted view of the operator. Upon the upper floor, with a northern exposure, are the microscopical rooms and the bacteriological laboratory, upon the southern side is the private room of the Superintendent. Between the two facing east is the library and study room of the hospital staff. This room is very large, excellently furnished; in short, complete in every detail. Standard medical works are contained in six large book-cases, engravings upon the walls, desks and arm chairs offer opportunity for work and ease. Beneath the tiers of the amphitheatre is the cloak room, from which the electric projecting apparatus is operated. Bausch and Lomb have

*South Front**East Front*

equipped the entire building with a complete outfit of chemical, microscopical and bacteriological apparatus, including a micro-photographic machine for which a special dark-room has been provided.

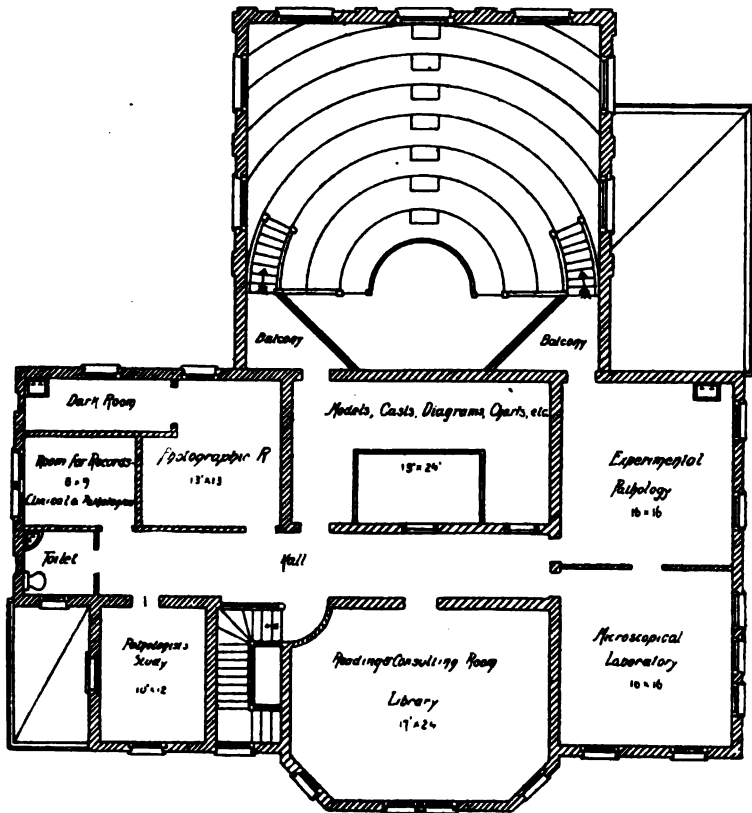
Altogether the new pathological institute is perfect, ranking far ahead of any similar structure in the United States or Europe which is



First floor

known to the writer, who has seen and worked in many of them. The many practical devices for chemical and microscopical work, the arrangements of the sources of water and light, and the magnificent work-tables and storing cupboards are really surprising to see, and most of them were made in the carpenter shops of the hospital itself.

It is the intention of the medical superintendent to maintain the department upon a high plane. Only original research will be carried on, notably into the causes and effects of mental and nervous diseases. Every case investigated will be clinically studied, so that the results of the chemical, bacteriological and pathological examinations will be rendered complete and valuable. Naturally some time must elapse before the department will be in smooth running order, or the researches be ready for close neurological and alienistic scrutiny. When this is the case, however, many important contributions to our knowledge of the etiology and pathology of insanity and the organic nervous diseases so frequently associated with various forms of mental affections may be hopefully awaited.

*Second Floor*

The Board of Control of the Central Hospital, viz., J. L. Carson, Pres., (Fairland); John Ostermann, Treas., (Indianapolis), and D. H. Davis, Secy., (Knightsville), deserves recognition for this effort to advance our opportunities for work and study in this branch of medicine. To the Superintendent of the Hospital, Dr. Geo. F. Edenharter, especial credit is due, for to his efforts, untiring zeal and constant care, the accomplishment of this high purpose is really due. He personally drew the plans of the building, devised the interior equipment, selected the mechanical and chemical apparatus, and watched the construction of the building. He is at the same time its engineer and founder, and in his capacity as medical chief of the hospital will govern the scientific working, clinical and pathological, of the new department. Institutions for the insane throughout the country will do well to follow the step taken at the Central State Hospital, and then we shall have abundant opportunity to work and study, and not, as is now the case, allow much of our material to go to waste. The establishment of similar departments of neuro-pathology will surely be followed by decided advancement in our still meagre knowledge of the causes of insanity and the organic tissue changes with which the mental affections are doubtless closely allied.

ALBERT E. STERNE, M. D.

Book Reviews.

VORLESUNGEN UEBER DEN BAU DER NERVÖSEN CENTRALORGANE DES MENSCHEN UND DER THIERE FÜR AERZTE UND STUDIRENDE VON PROF. DR. LUDWIG EDINGER. Fünfte stark vermehrte Auflage. Leipzig, 1896. (Lectures on the Structure of the Central Nervous System, etc. Fifth edition).

The fifth edition of Edinger's now famous lectures on the structure of the brain and of the spinal cord in man and animals marks an advanced stage in the evolution of a great work. Did we not know the author's untiring energy, and the popularity of his book, we might suppose that the terminal stage had been reached. But further editions will be demanded and, judging by the character of the past issues, each one will be a distinct improvement upon its predecessors. The rapid progress made in this special department is due in no small degree to Edinger's original researches. While he does not exhibit any undue haste in incorporating newly discovered facts with those that have become common property, he does not hesitate to introduce observations which bear the *prima facie* evidence of being correct. His own line of work makes Edinger an excellent critic of the labors of others and renders his book quite as authoritative as that other great German work, Kölliker's *Gewebelehre*, is in the same and kindred fields.

It has been the author's special aim in this new edition to elucidate the architecture of the central nervous system by a study of its various parts in the lower animals, and after showing the manner of development and the full grown condition of the nervous system in the lower animal series, he leads up to a clear understanding of the complex nervous system of man.

Edinger's lectures constitute, practically, a comparative anatomy and embryology of the nervous system.

Part II of the present book contains the new and important chapters. In studying the lower vertebrate series, the author is able to discriminate between the fundamental and the accessory parts of each division of the central nervous system, and by the recurrence of fundamental tracts is able to prove the dependence of elementary functions upon those structures which are present in an entire series, from the lowest to the highest. The author's method is well illustrated in the chapter on the construction ("Aufbau") of the spinal cord. He shows very clearly, by reference to the spinal cords of the lower vertebrates, how the posterior grey columns (they are not horns) result from the entrance into the cord of the sensory nerve fibres, and how the size of these columns is entirely proportionate to the number of fibres received into the cord. The anterior columns have a similar relation to the motor fibres and vary in closely related animals in proportion to the development of the extremities.

After discussing the parts which go to make the spinal cord an independent centre, for such it is, even in higher vertebrates, the author traces the tracts connecting the spinal cord with other parts of the central nervous system—chiefly with the cerebellum, with the interbrain and the mid-brain. Each point is aptly illustrated and brought out in

the author's remarkably lucid style. In the twelve pages of Part II., devoted to the structure of the spinal cord, there is more rational information than in the lengthy topographical description of the older text books. Each part of the brain is presented with equal care and with the same wealth of illustrations: but what may be expected from Edinger's comparative embryological method is exemplified best in the chapter on the diencephalon. And why should we not refer to this method of studying the central nervous system as the "method of Edinger"? It promises, in the near future, even better results than have been reached by the methods of Flechsig and of v. Gudden.

Of Parts I. and III. the reviewer will say little, for they contain the main chapters of former editions, though much new matter and many new illustrations have been added. We note with regret a single omission. The appendix to the fourth edition, on the newer methods to be used in researches on the structure of the central nervous system, should have been included in the fifth edition also, for the book in its present garb will act as a stimulus to earnest students who would welcome a concise chapter on "technique." The topographical work is beyond praise.

We are informed that an English translation of the fifth edition is soon to appear. It is to be hoped that Dr. Vittum, who gave a very acceptable rendering of the first edition, will make the book worthy of the original.

B. S.

NOTHNAGEL'S SPECIELLE PATHOLOGIE UND THERAPIE. DIE GE-
SCHWÜLSTE DES GEHIRNS von Prof. Dr. H. OPPENHEIM, in Berlin.
Vienna, 1896. (Tumors of the Brain).

Twenty years have passed since the publication of Ziemssen's Encyclopaedia. During this score of years such remarkable progress has been made that there is every warrant for the issuance of this new work. Prof. Nothnagel has shown excellent judgment in "dividing parts," but such judgment is dependent in the first instance upon an intimate acquaintance with the entire medical literature of the day. It is unfortunate that similar works have been planned by men of inferior learning, who have been guilty of all sorts of absurdities in assigning subjects to men whose past experience was not such as to lend authority to their opinions. A more minute subdivision of subjects than in Ziemssen's Encyclopaedia will make Nothnagel's series a collection of most valuable monographs. Neurology has fared particularly well. Leyden and Goldscheider are at work on the diseases of the spinal cord and medulla oblongata; Bernhardt has written a very exhaustive treatise on the peripheral nerves, Ewald one on myxœdema and kindred affections and Moebius on exophthalmic goitre. Krafft-Ebing has treated of dementia paralytica, and Oppenheim has done excellent work in discussing syphilis of the nervous system and tumors of the brain. To the last-named monograph we desire to call especial attention to-day, but we hope in the course of the next few months to do justice to some of the other monographs in the series.

Oppenheim has discussed tumors of the brain with great care. There is evidence everywhere of a thorough acquaintance with the German, French, English and American publications, and of a wide personal experience. We are glad to see that the author prefers a careful weighing of results in a few and well-observed cases to the array of meaningless statistics. This applies particularly to the discussion of the symptomatology and of the results of operative procedures. The author has adopted the plan of making general statements and of adding in parenthesis the names of those authors whose writings and opinions supply the evidence. By reference to the literature at the end of this volume

any statement can easily be verified. Each symptom, headache, optic neuritis, convulsions, etc., is discussed fully. There is no room for criticism, as the reviewer finds himself in thorough accord with the author after a very careful study of his monograph. In the chapter on differential diagnosis attention is directed to the fact that double optic neuritis may occur in association with other conditions, such as acute infectious diseases, intoxications, multiple neuritis, etc. Its occurrence as a symptom of syphilis has not been sufficiently insisted upon. In passing we may note that Oppenheim does not believe that the tendency of patients to fall to one side has any localizing value in tumors of the cerebellum; nor does he believe that cerebellar ataxia points to involvement of the vermis, and not of the cerebellar hemispheres. With the latter view we are bound to agree; the former cannot be accepted without reserve.

Operative procedures for the removal of tumors have the sanction of the writer. Oppenheim weighs all the facts to be deduced from the cases hitherto reported, and believes there is much to be gained by operation at the proper time and in the proper region. He does not consider that the size of a tumor need cause one to decide against the practicability of an operation, as the results of Keen, Braman, and Erb have shown. He says little about the necessity of controlling hemorrhages in all these operations. This is left properly enough in the hands of the surgeon, but our own experience has taught us that there is in all cranial operations one paramount danger, and that is the loss of blood. Why have surgeons so long delayed the plan of applying a firm circular ligature? We are quite in accord with the view that in every case the patient should be given the benefit of medicinal treatment before an operation is attempted. A period of six weeks would seem to be quite long enough for such a trial; if delayed beyond this, valuable time may be lost. Since cases that have been improved by drugs are altogether exceptional, we believe that the advisability of operation may be entertained as soon as the diagnosis of an operable tumor has been established. Bruns is right in maintaining that every operation for cerebral tumors is an exploratory operation, and should be urged as such upon the patient. We subscribe also to Oppenheim's opinion that for the present cerebellar tumors belong to the category of inoperable cases. Lumbar puncture is advocated as a palliative measure in certain cases. The danger of this procedure in cerebellar tumors should be made generally known. The present writer's experience leads him to endorse Fürbringer's opinion.

Oppenheim, and for that matter Bruns also (in Eulenbarg's *Encyclopædia*) have reviewed the entire subject of brain tumors so thoroughly that future writers need not analyze the antecedent literature, but there is need of further careful reports on the surgical treatment of brain tumors.

B. S.

Correspondence.

Dr. Lewellys F. Baker, Associate Professor of Anatomy, Johns Hopkins University sends the following letter concerning neurological nomenclature.

To the Editors:—

The nervous system as is well known, was formerly described as being made up of nerve cells and nerve fibres. Each peripheral fibre of the cerebro-spinal system consists of an axis cylinder around which is a fatty sheath, and outside this again is another sheath, the neurilemma. Bundles made up of great numbers of these nerve fibres held together by firm fibrous tissue run through the tissues of the body and are known as "nerves." The term "nerve" as originally employed, has reference to the firm, sinewy or tendinous character, (Latin *nervus*; Greek *neuron*), of these bundles, a quality dependent in reality on the fibrous connective tissue of the bundle rather than upon the really functioning irritable structures within it. The terms "nerve" and "nervous" are now connected in thought rather with the functionally irritable structures.

The relations of the nerve cells to the nerve fibres remained for a long time unknown. The nerve cells occurring in groups within the nerve centres were known to possess branched processes, the so-called protoplasmic processes or *dendrites*. Later on it was shown that the axis cylinder of every fibre is always a process, an integral part, therefore, of a nerve cell, though this process is very different in form and probably in function from the other processes (*dendrites*). It has recently been demonstrated that the whole nervous system is made up of units, each unit consisting, as a rule, of a nucleated cell body with its *dendrites* together with one or more axis cylinder processes with side-branches (*side-fibrils* and *collaterals* or *paraxones*) and end-ramifications. Each of these units inclusive of all its processes is in reality a single cell of the body, quite analogous to a single liver cell, or a single muscle cell, and a very suitable name for the unit would be "nerve cell," were it not for the fact that this term has been used for decades to indicate only a portion of the unit as mentioned above, namely, all except the axis cylinder, and in many minds would call forth this erroneous idea.

It remained, therefore, to find a satisfactory name for the whole nerve unit. Waldeyer of Berlin, suggested that from the Greek *ho neuroon* a new German word be coined *der Neuron* (*pl die Neurönen*), and the introduction of this term has been of significant influence in making the ideas, involved in what is now generally termed the *neurone-conception* of the nervous system, generally known and appreciated. The term has been in Germany almost universally adopted by morphologists, histologists, physiologists and clinicians notwithstanding the objection offered by v. Kölliker that the term *neuroon* in reality indicates "einen Sammelpunkt vieler Neurönen oder Nerven." He has suggested that the word *Neurodendren* or *Neurodendridien* be used instead. Van Gehuchten has adopted Waldeyer's word spelling in French "*le neurone*" and French writers generally employ it. The leading investigators in

Spain and Italy have also adopted the same term; so that even if it were etymologically somewhat objectionable, its use has become so general and cosmopolitan that it seems as though we must also employ it in English. Baker's suggestion that we use the term *neure* is a very good one, but the term of Waldeyer has already become too prevalent to be easily supplanted. The question arises, how is Waldeyer's term to be anglicized? Would it be justifiable to bring it into English through the French and to spell it *neurone*, pronounced *neurone*, or could it be brought into English directly from the Greek and be so spelled and pronounced? It is especially desirable that this spelling and pronunciation be permissible owing to the fact that a few writers, among others, Schaefer and Donaldson have employed another word *neuron* (Greek *neuron*) to mean the axis cylinder process, a nomenclature which is obviously etymologically faulty and which in my opinion is not likely to become generally popular owing (1) to the existence of a better term for the axis cylinder, viz., *axone* or *neuraxone* (Greek, *Xoon*) already current; and (2) to the likelihood of its confusion with the word introduced by Waldeyer for the whole nerve unit, a word now in universal use in other countries.

For the sake of avoidance of confusion in the bibliography a speedy agreement concerning the nomenclature is certainly highly desirable. I have submitted the question, very much as outlined above, to Prof. B. L. Gildersleeve of the Johns Hopkins University, with a request for aid, which has been courteously and promptly given. Prof. Gildersleeve writes me that v. Kölliker's objection to *neuroon* will not hold, for it would apply equally as well to *parthenone* which means "the House of the Virgin." He adds "while the spelling *neurone* is not pleasing, still for that matter the spellings *anode* and *cathode* are just as objectionable, since after the analogy of *method* they should be spelled *anod* and *cathod* and under the circumstances *neurone* seems to be inevitable." It is a matter of congratulation, I think, that neurologists may thus use the term in English with the sanction of a recognized authority on Greek. Etymology if medical and scientific writers will co-operate, we may finally hope to bring about the establishment and maintenance of a uniform international nomenclature.

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Original Articles.

GENERAL PARALYSIS IN TWO SISTERS, COMMENCING AT THE AGE OF TEN AND FIFTEEN RESPECTIVELY.—AUTOPSY IN ONE CASE.¹

By AUGUST HOCH, M.D.,

Assistant Physician and Pathologist, McLean Hospital.

GENERAL paralysis in early life, i. e., under the age of twenty, has, so far as I can see from the literature, not been reported in this country; in fact, altogether I can find no more than about fifty cases, among which certainly thirty-five have been described since the year 1890, and over twenty in the last two years. Among all the cases about the same number, i. e., twenty, have been described by English and German observers. About seven cases are found in the French and a few in the Russian literature.²

The two cases to be reported may, therefore, prove of interest, particularly since they represent the occurrence of the disease in two members of the same family, particularly, also, since the autopsy in one of our cases showed changes which by themselves I consider of special interest.

¹From the laboratory of the McLean Hospital, Waverly, Mass.

²For a complete bibliography up to 1895, see article of Alzheimer: "Die Frühformen der allgemeinen progressiven Paralyse."—Allgemeine Zeitschrift für Psychiatrie, etc., Vol. LII., page 533.

The cases are typical, and by the examination of the nervous system the diagnosis is raised above all doubts. The younger sister I have not observed myself. She was under the care of Dr. Cushing, in Haverhill, who furnished me with some information; the bulk of the history, however, I got by collecting the data from the different members of the family whom I went to see. The clinical picture can, therefore, not be expected to be in any way complete, and the following description will only give in a very general way the course of the disease.

Case I.—*Ida*. The patient was healthy as a baby and, according to the statement of different members of her family, in every way normal. She was even considered a very bright child, whereas her sister, whose case is to be spoken of later, is said to have been of moderate average intelligence. The time of onset of her disease is difficult to fix and was certainly very gradual, but when the patient was about ten years old it was thought that she was growing lazy. Instead of playing with other children she would sit around the house in an apathetic way. She frequently complained of pain about her stomach, but had no vomiting. On account of this trouble she is said to have been kept out of school. When about 11 years of age she began to have marked motor symptoms, staggered about a good deal "like a drunken man," and on that account she had to be guided around. One of my informants, her sister, expressed the course of the disease with reference to the motor symptoms in short as follows: "Two years we dragged her around, two years she sat in a chair and two years she was in bed." From about fourteen years the patient's legs had a tendency to be drawn up, and while the patient was in bed in the latter part of her life this became so marked that even passive extension was difficult. Later a similar condition appeared in the arms and toward the end they were drawn over the chest. For the last two years it was impossible for the patient to sit up in a chair. The voice was noticed to be coarser than it had been and the speech to be indistinct; towards the end of the patient's life it was nothing but a mumble. In the meantime the mental condition approached more and more a complete dementia, and for about a year before death the patient was so feeble-minded that she knew no one. She lay in bed in the

above described contracted condition, untidy, mumbling, picking at her bed-clothes, unable to help herself in any way. The course was apparently one of progressive dementia without any excitement or any delusions. At no time during the course were any convulsions noted until shortly before death when she had a series of them. She died in convulsions a week after their onset.

The autopsy was made by Dr. E. W. Taylor in the presence of Dr. Putnam. To the former I am indebted for the description of the gross appearances of the nervous system; to Dr. Putnam it is my privilege here to express my indebtedness, not only for the pathological material which he put at my disposal, but, also, for drawing my attention to these cases. It was with him that I first saw the sister who is now living. In this place I would also express my thanks to Dr. Cowles for his kindness in admitting the latter to this hospital.

Before giving the anatomical details gained from a study of the tissues, I wish to give an abstract of the clinical notes made on the sister whom we had in the hospital, as well as give some data with reference to the family history.

Case II.—Rosa, now nineteen and a half years old, has been a healthy child and of average intelligence up to the onset of the present disease; for the latter there is no cause to be made out. From her fifteenth year on she got along badly in school, and since about her sixteenth year she has been, even according to the judgment of her family, feeble minded. She has never had any excitement nor any delusions or hallucinations. Her mental weakness was first shown in an inability to follow school work, then in lack of interest in anything, in a certain childishness, in a loss of memory and, finally, in an inability to understand even the simplest things. To give an example: she has for the last few years very frequently done crochet work, always using the same stitch; the most painstaking attempts on the part of her sister to teach her even the slightest modification of this were fruitless. For two or three years her gait has been changed and she is somewhat uncertain. Her speech also became altered, the change coming on gradually. She has had no convulsions. This is, in short, the history up to the time I saw her. The observations which on frequent occasions were

made during her stay in the hospital, i. e., during about five months may be summed up as follows (to give a résumé of the notes made is particularly easy since the change has not been great; no new symptoms have appeared, but she has become somewhat more demented, her walk more uncertain, her tremors more marked): She is distinctly feeble-minded; after her arrival, although she had been told that she was coming to this hospital, she was not clear where she was; she was satisfied at once and when her sister left her she did not mind it. Afterwards she frequently cried and said she was homesick, but a few words completely changed her feelings, which were evidently but very superficial. In spite of being in a rather noisy ward, associated with persons with many marked peculiarities, this did not disturb her or make any impression upon her; in other words she was, although quiet and orderly, absolutely unable to appreciate her surroundings. A picture was shown to her, and her description of it, which she was asked to give, was very elementary and superficial; her behavior was often silly. During an examination she would often tell of a perfectly irrelevant incident which had happened in her neighborhood, or laugh convulsively. She was able to recite fairly well a number of poems she had learned when in school, but it was evident she had hardly grasped the meaning of them. Thus it happened that she left out lines and words without noticing it, making the meaning incomprehensible. On several occasions the patient was asked to read, and the results here obtained were quite characteristic. From a number of tests made on different occasions I shall only give a general abstract. She was asked to read a paragraph from "Alice in Wonderland." She made mistakes like the following: Instead of unfortunate she read uniform; for execution, express; for sneezing, snize; for choking, colking; for lizzard, laze, and other similar errors; while many words were also read correctly. Individual letters could always, smaller words almost always, be correctly read when shown alone, sometimes only after spelling, e. g., mock, fashion. Longer words, like squeaking or even turtle, could not be read, even after spelling; in the same way words which probably she had never heard or seen, like gryphon or guinea pig, could be spelled, but no attempt was made to pronounce

them. Figures could be read alone or two or three together, frequently even four, but never more. Writing was difficult to obtain, since the patient could not be made to write much, but the few specimens show not only mental defects analogous to those expressed in reading, but, also, very marked motor disturbances, i. e., tremulousness. To anyone familiar with the subject her handwriting would at once have suggested general paralysis. She calculated very badly and, with the exception of the smallest multiplications, with two or three, possibly four, she did not give correct answers. Multiplication she did better than addition or division. This is evidently due to the fact that the multiplication tables have by practice become more fixed associations than the additions or divisions. She evidently depended entirely on some well-rooted associations, for where examples with any higher figures, as e. g., 5×6 or $13-11$, were given to her, she not only did not do them at once, but knew no means of getting at the results; in other words, no matter how long a time was given to her to solve the problem she never reached a solution. Quite a large number of tests were made, and the patient, both of her own accord and with the assistance of the nurse, attempted frequently to write down the multiplication tables. She improved her calculation hardly any by this practice. It should be emphasized here once more that the patient was in school up to her fifteenth year and had been perfectly able to read, write and calculate. Rieger and his pupils have pointed out that disturbances like these, if at all pronounced, are very characteristic of the dementia of general paralysis.

The gait was markedly abnormal. It was tottering, the legs placed far apart and held somewhat stiffly; the feet came down on that account somewhat as in a flat-footed person. She wavered much from a straight line. The arms, during walking, were held away from the body as if for balancing the latter. She can stand with her eyes closed and feet close together, but cannot stand on one foot at all. The speech is indistinct, monotonous, high-pitched, and has a certain vibration to it; frequently words are somewhat slurred over; at the same time there is a coarse tremor about the mouth and chin and a similar tremor is seen in the tongue. The face is somewhat one-sided; this is seen at rest as well as in motion. The patel-

lar reflexes have been throughout markedly exaggerated and there has been on different occasions a more or less well marked patellar clonus but only a slight ankle clonus. The reflexes in the arms were increased, particularly during the latter part of her stay in the hospital. The pupils have been the same all along. The right was larger than the left on every examination, and neither eye reacts to light or accommodation.

The condition of the sensibility is of interest. She perceives tactile impressions very well, but there is marked diminution in her appreciation of pain sensations. At first she not rarely failed to recognize the pin as such; when her attention was drawn to it she frequently differentiated the head and the point of the pin correctly, but she never minded the pain; in fact, a fold of skin could be transfixed without her wincing. The same thing was seen in applying the faradic or galvanic current of full strength; it was borne without pain, even if the metal of the electrode was directly applied.

The patient shows no signs of hereditary syphilis. The upper teeth are badly decayed, those left are not Hutchinsonian, the corneae are clear, the shins smooth, the liverdulness normal.

So much for the clinical history of the second patient.

Family History—These two girls are the youngest members of a family of 7 children. The others are healthy and normal. None show signs of hereditary syphilis. None have died except Ida. The mother had no miscarriages.

The father, a Dane, has been unable to work for about twelve years, although he is now only sixty-one years old. He frequently became dizzy and was said to be unable to direct his hands properly. Six years ago he had an attack in which he got pale, fell down and was weak on one side. He recovered completely. He now falls not infrequently, partly, as he says, because he becomes dizzy, partly because he stumbles. He is said to have had frequently sores break out over his body, and last year two "boils" appeared on the forehead, which were present most of the summer; they finally broke open and dead bone was found in them. At the same time he had a good deal of headache. He is irritable, but has been so for years. Never does any work, but sits about the house, perhaps reading

in his old Danish Bible. He denies positively ever to have had any syphilis. On examining the man I found him looking certainly older than he was and quite infirm. There was a tremor of the head from side to side, a tremor of the muscles about the face when he spoke, but the speech was not altered. There was a certain amount of difference in the two sides of the face. Tongue protruded straight and did not tremble. He has some tremor of the hands and the handwriting is tremulous. The muscular strength of the arms and legs is fair and equal on the two sides. The gait is very infirm and tottering, but not ataxic; it became worse when his eyes were closed, but he stood very well with his eyes closed. The knee-jerks are diminished. No changes are seen about the eyes, the pupils react to light and distance. On the forehead there are two deep ragged scars and there is evidently loss of bone. His arteries are somewhat thickened.

I do not see that we can make a definite diagnosis in this patient; he certainly has not general paralysis. The most likely explanation seems to me—to be sure after too short a period of observation—that the man is prematurely senile; he has a certain amount of arteriosclerosis. That the man had syphilis is made probable by the appearance of the two “boils” on the forehead.

The mother, also, is infirm and complains of all sorts of pains. She dates her troubles from the sickness of her daughter, whom she nursed. Examination was negative. She is intelligent and seems merely nervous and hysterical.

Autopsy on Case No. I—(Dr. Taylor)—Dec. 1, 1894. Girl sixteen years old. Size of body, that of a child of ten years of age; body emaciated. Lower legs particularly thin and emaciated in comparison to the body, where there is considerable subcutaneous fat. The head is well formed; no deformity.

Slight erosion of the inner table of the skull on the left side; normal thickness of skull. Dura mater is normal. Considerable serous fluid runs out on removal of the skull-cap. The pia mater is opaque and milky in appearance, especially over the sulci. The brain substance is firm on pressure. The convolutions are very pale and bloodless and the substance firm on section. The cortex is considerably thinner than normal. There is considerable fluid in the ventricles. The pons, medulla and spinal cord show no abnormality macroscopically.

The internal organs are normal. The uterus is very undeveloped.

Microscopical Examination.—Microscopically there were examined different sections of the brain cortex, portions of the cerebellum of the basal ganglia and different levels of the pons, medulla and spinal cord.

The cortex shows the changes commonly found in general paralysis. The pia is thickened and there is great infiltration with lymphoid cells. With reference to the ganglionic cells little can be said. The tissues were hardened in Müllers fluid, but the cells were not well preserved. With a low power the most striking feature in the cortex everywhere is the meshy appearance in many places, which is most marked in the most superficial layer. We note also wide spaces around the blood vessels and around nerve cells, the apparently increased number of nuclei throughout the tissue and the great infiltration of the adventitial spaces with lymphoid cells. All the vessels are very conspicuous on this account. With the high power the most striking feature is the large number of coarse spider cells, which are very evident in the superficial and deep portions of the cortex, especially in the lower portions they are more pronounced than is commonly seen in general paralysis. They are often very coarse and can be distinctly seen with a low power. The bodies of these cells are often quite large and stained deeply with fuchsin. In the white matter also spider cells are seen and, as it seems, they are more numerous the nearer one gets to the cortex.

The medullated nerve-fibres appear much diminished, not only the tangential fibres in the superficial layer which with the exception of a few places in the occipital lobes, are almost entirely lost, but, also, the horizontal fibres lower down and even the radial. This diminution is very great and very widespread. It may be due to the staining method, but, on the one hand, the change was well marked in the coarser fibres, and, on the other hand, the fibres in the white matter were well stained, although distinctly diminished in many places. It should also be remembered that the patient began to become affected with the disease very early in life, at a time when the cortex

³The tissues were stained by Weigert's method and with Alum-Haematoxylin and a dilute watery solution of Fuchsin.

does not show all the fibres which it has in mature life.⁴ The changes on the whole were very pronounced and well marked all over the cortex, although less marked in the posterior than in the anterior regions.

In sections from the optic thalamus very similar changes were seen as in the cortex; here, also, large, sometimes huge, spider-cells were found, and the vessels showed large accumulations of lymphoid cells. The latter condition, in fact, was seen all through the nervous system, no matter what portion was looked at. But, aside from this, the vessel walls looked normal with the exception of a few places in the basal ganglia, where there was distinct hyaline degeneration of vessels and where amyloid bodies were seen.

In the cerebellum the pia also showed thickening and marked infiltration with lymphoid cells. The changes in the cortex are interesting. In places it is perfectly normal, the Purkinje cells well formed with well stained nuclei, the layer above them homogeneous looking. In other places there are marked changes, the cells look hyaline, deeply stained with fuchsin with ragged outlines, misshapen, and with nuclei that stained very deeply and evenly and which presented irregular outlines. These changes were seen in different regions in different degrees and in places the Purkinje cells had completely disappeared. Where these alterations are seen the entire cortical substance is much changed; it has a fibrillary appearance instead of the normal homogeneous one, the fibres are coarse and chiefly directed vertically, although many are oblique and horizontal, the latter particularly in the most superficial portion where they form a dense layer. Well marked spider cells are seen, especially in the most profoundly altered portions, and the whole thickness of the cortex is more or less reduced. We have here a great increase of neuroglia, which has, no doubt, followed the degeneration of the Purkinje cells with their great branching systems.

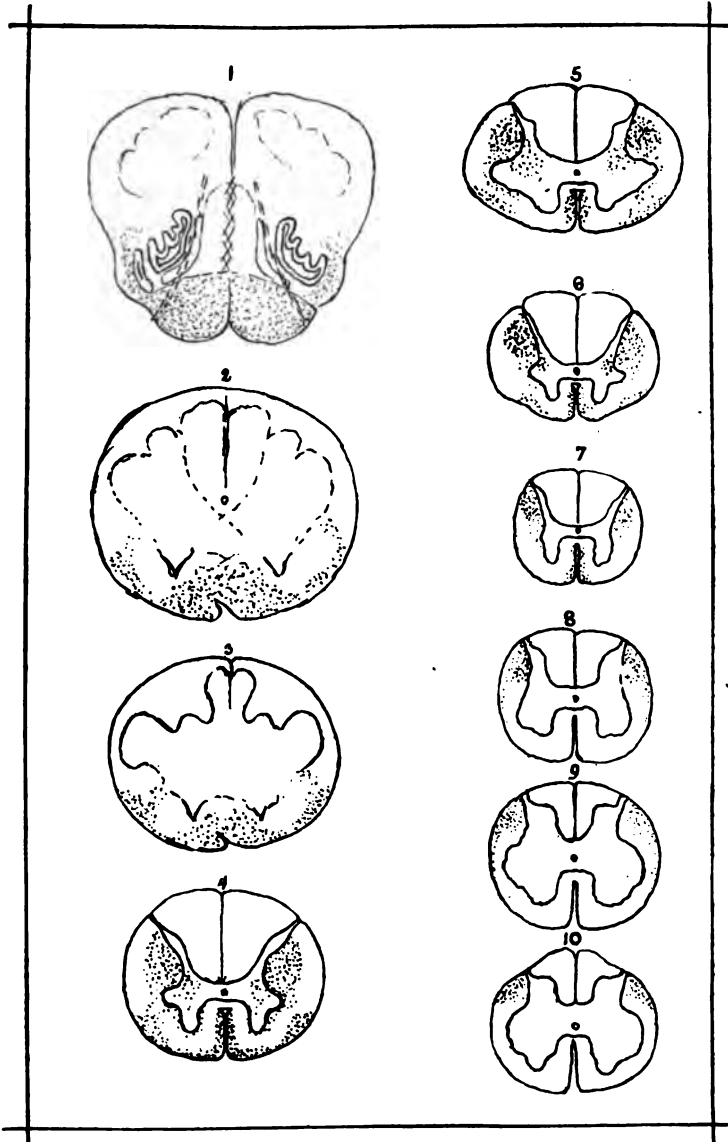
The uneven distribution of the changes is quite striking, and it is of interest to note that the affected parts are

⁴Cf. Vulpius: Ueber die Entwicklung und Ausbreitung der Tangentialfasern des menschlichen Grosshirns während verschiedener Altersperioden. Arch. f. Psychiatrie. Band xxiii (1892), p. 775.

chiefly in the periphery of the organ, whereas the deeper portions are usually normal.

Spinal Cord.—In the sacral cord there is degeneration, chiefly confined to the lateral pyramidal tracts, although it extends evidently also somewhat inward (Fig. 10).

In the lumbar cord the same condition is found, namely, the degeneration of the lateral pyramidal tracts; here it extends, perhaps, somewhat ventrally along the periphery of the cord. As we ascend, the field of degeneration approaches nearer to the gray matter, whereas at the periphery it is less pronounced, this corresponds again to the position of the lateral pyramids, while there is also a certain amount of degeneration along the margin (Fig. 8). In the dorsal cord we begin to see with certainty a degeneration in the field of the anterior pyramidal tracts; the light appearance in the lateral columns (in Weigert sections) is well marked, but not quite confined to the pyramidal tracts; it extends forward, although it is better marked in the former region. It must be stated that at no place is there anything like a complete degeneration in the lateral pyramidal tracts, but the changes in a Weigert section are clearly enough marked to be very well seen with the naked eye. The field of the direct cerebellar and of Gowers' tracts is entirely free from degeneration. In the upper dorsal cord (Fig. 6) the direct cerebellar tract is well stained, but ventrally from it there is a distinctly lighter appearance, i. e., in the region of Gowers' tract. The degeneration in the lateral columns is well pronounced in the region of the pyramids, but shades off in a ventral direction and blends with the degeneration in the region of Gowers' tract; it also extends along the outer surface of the posterior horns and the pyramids degeneration is present, but not so well marked as in the region of the pyramids; so that in this direction also the degeneration is not quite confined to the lateral pyramids, although, again, it is more pronounced here. The anterior pyramids show distinct changes, particularly on one side, but these do not extend along the anterior border of the cord. Very similar is the condition in the cervical cord. The direct cerebellar tract is perfectly normal, the pyramidal degeneration shading off in a ventral direction and around the an-



DESCRIPTION OF THE PLATE.

The black dots indicate the regions of degeneration; in the gray matter of the spinal cord the dots indicate the great development of spider cells.

terior horns, blending with the degeneration in the region of Gowers' tract, which has now become more pronounced. The anterior pyramids show here also more pronounced degeneration. As we go up still higher (Fig. 4), (the level of the accessorius), we find similar conditions. Here, however, there is a field of degeneration in the region of Gowers' tract which is more circumscribed and more pronounced than in any region below; it extends in a triangular projection towards the anterior horn and does not extend dorsally as far as the outgoing root of the accessorius, but only to a point which is about on a line with the ventral border of the lateral projection of the gray matter; interesting is also the fact that it extends inward and ventrally to the field of degeneration in the anterior pyramids, which here shows, unlike that lower down, a marked extension outward. The connection between this and Gowers' tract is well marked, but the degeneration here is only a small strip. It is necessary to state here that, unfortunately, the different levels were not marked, and that a large part of the cervical cord could not be examined, so that it is impossible to say where this more pronounced field of degeneration began.

As we ascend from here into the medulla, at a place where the nucleus of Goll is well formed, but the pyramidal decussation not yet finished (Fig. 3) we find the degeneration in the field of Gowers' tract well marked and having about the same place as lower down; it is connected by a strip with the degeneration in the pyramids, a strip which is distinctly separated from the periphery by normal-looking nerve fibres. There is also a slight degeneration extending from this well-marked field slightly dorsally and inward to the anterior horns. Still higher up, where both the nucleus gracilis and cuneatus are well formed, at the level of the sensory decussation, the degeneration is still in the same position, separated from the periphery by some longitudinally cut fibres and blending with that of the pyramids, which is still well marked; here, also, it extends inward. Still higher up, where the olive appears, we see in Weigert sections that the field adjoining the pyramids, namely, the region around the olive inside of the external arciform fibres, is considerably lighter than normal and in the angle just ventral to this region there is a somewhat lighter appearance than normal. Above this

region, i. e., where the olive is well formed, the degeneration cannot be made out with certainty. The field situated in the angle at the external and dorsal side of the olive is light, but it is also so normally, so that a slight degeneration could probably anyhow not be made out easily, in Weigert sections, nor was it any more evident in fuchsin preparations. The pyramids are still seen to be partly degenerated. This latter degeneration can be followed through the medulla and partly through the pons; in the higher portion of the latter it becomes questionable, and in the crura cerebri it can no longer be made out.

The Gray Matter—There are here and there ganglionic cells in the anterior horns which are with certainty changed, having the same hyaline character as seen in the Purkinje cells described above; these changes are, perhaps, more marked in the dorsal cord. In the nuclei of the medulla and pons similar cells are found, but pronounced degeneration is found in none. As we stated above, the infiltration with lymphoid cells around the vessels is seen here as well as in other portions of the central nervous system. This is particularly well marked in some vessels in the pons. Another important alteration to be noted is the very marked changes in the gray matter of the spinal cord. We find in sections from the upper dorsal and the cervical cord a marked growth of neuroglia, which is seen in the appearance of numerous well-pronounced coarse spider cells which can be seen even with a low power. These cells are chiefly seen at the base of the anterior and posterior horns and in the centre of the gray matter on either sides, although isolated large neuroglia cells are also seen in other places. In the lower regions of the spinal cord this feature is not present. In the nerve roots as far as they were examined (those which were attached to the spinal cord sections) no changes were found.

Remarks on the Pathological Findings.—The changes in this case are very profound. They are well marked, not only in the cerebral cortex, but also in the basal ganglia, in the cortex of the cerebellum, and in the white and gray matter of the spinal cord and the medulla. With reference to the spinal cord the question arises how to interpret the changes which we have described. In the lateral as well as in the anterior columns we have seen a partial degeneration, confined in the latter to the ante-

rior pyramids. Degeneration in the lateral columns is not an infrequent occurrence in general paralysis as is well known; it has of late been attributed by Pierre Marie in some cases to changes in the gray matter of the cord which gives rise to a degeneration of tract cells, and thus to loss of short tracts in this region. As reasons for this assumption the facts have been adduced that the degeneration is not confined to the lateral pyramids and is not present in the anterior pyramids. In our sections, however, we find throughout the entire length of the cord a degeneration which in some places is entirely confined to the lateral pyramids; in other places, though not limited, is certainly best marked in this region; besides it diminishes downward, *pari passu*, with the diminution of the pyramidal tracts. Moreover, we find a degeneration in the anterior pyramids which we can distinctly follow down the dorsal cord. All these are features which make the degeneration look like a secondary one due to cerebral changes; at any rate it is fair to say that we have before us a systemic degeneration in the central motor neurons, although the changes in the nerve fibres surrounding the pyramids must be attributed to degeneration of short tracts; in other words, the latter are not merely an extension of the process into the surrounding tissue, but are probably due to alterations in the gray matter of the cord. Whether, in spite of the appearances in the crura, there is a degeneration which is not evident in Weigert sections, or whether the process becomes really completely lost in the upper pons, is difficult to say. It is, however, possible that, as has been suggested by others, only the most peripheral portion of the neuron becomes first affected owing to its distance from its trophic centre and that gradually the process extends nearer to the latter. With gradual chronic changes and a slow degeneration of nerve cells as is the case in general paralysis this view is not improbable. I am inclined to see, therefore, in this degeneration of the pyramids a secondary effect of changes in the cortex.

With reference to the degenerated areas in the antero-lateral region of the cord, there can hardly be any question but that we are dealing with degenerated fibres in Gowers' tract. It is noteworthy that this only appears in the upper portions of the cord, more particularly in the

cervical region, becoming more pronounced as we ascend and very well marked in the upper cervical cord and the lower portions of the medulla. It cannot, however, be followed far up in the medulla; but this need not surprise us, because the fibres of Gowers' tract become rather scattered, and it would be difficult to see a partial degeneration in Weigert sections.

In view of the fact that Hoche⁵ has demonstrated an anterior portion of Gowers' tract which extends to and along the anterior fissure, the question must naturally arise whether, perhaps, the degeneration along the anterior fissure and that in the antero-lateral region do not both belong to Gowers' tract; on the other hand, the fact that this anterior degeneration extends well into the dorsal cord a long distance below the antero-lateral degeneration and the fact that the degeneration of the pyramids is well marked in the medulla oblongata, i. e., before the separation of the anterior and lateral pyramids has taken place, speak against this, although it is not excluded that a part of the degeneration field really represents fibres belonging to the antero-lateral tract; indeed, this seems rather likely. High up in the cervical cord the degeneration in the antero-lateral region is continuous by a fine strip with the degeneration in the anterior pyramids and in the medulla the same is true, here particularly the degeneration extends further ventrally than the degeneration in Gowers' tract usually does as we observe it after sections of the cord. In one of Hoche's cases, however, where the lesion was high up (I. D.) the degeneration also extends further anteriorly. It is possible that the higher the lesion the further forward does the degeneration extend. Whether the well-marked loss of fibres around the olive represents in part at least fibres from Gowers' tract or whether these are entirely different fibres is difficult to decide. The question which next arises is, What accounts for these degenerated fibres in Gowers' tract. According to Lenhossék⁶ it is "probable" that the cells which give rise to the fibres of Gowers' tract are situated "partly in the centre of the

⁵Cf. Hoche: Ueber secundäre Degeneration, etc. Arch. f. Psych., xxviii., Heft 2, p. 517.

⁶Der feinere Bau des Nervensystems im Lichte neuester Forschungen, 1895. p. 408.

anterior horns, partly in the central zone of the gray matter." We have seen that in the upper portions of the dorsal, more particularly in the cervical cord and here increasing as we advance upward, changes are found at the base of the anterior and posterior horns and in the centre of the gray matter on either side, indicated by a very marked increase of neuroglia. While to these changes we must probably in part attribute the degeneration in the ground bundles, it seems fair to suggest that the degeneration in Gowers' tract is also due to the destruction of cells in this region.

Another feature of interest in these cases is the fact that two members of the same family were affected at this early age with this disease. I have been able to find this in only one other instance in the literature. These cases, reported by Justschenko,⁷ are a brother and a sister, who were attacked by the disease at the age of fourteen and fifteen respectively. Both patients had signs of hereditary syphilis. Cases like these are qualified more than any others, it seems to me, to show how untenable is the idea that general paralysis is due to exhaustion and point to the rôle which hereditary influences play in the production of general paralysis at such an early age. It has been shown by Alzheimer,⁸ who has collected forty-one cases of juvenile general paralysis, among them three of his own, that there was a neuropathic heredity in about seventy per cent., but the heredity which seems most important in these cases is syphilis. The same author finds that fifty per cent. had with certainty a syphilitic heredity; if those were added who had very probably such antecedents, the figures would rise to 85 per cent., and if still those were added in whom a syphilitic heredity was from certain data probable, to ninety-one per cent., an unusually high percentage. It is, of course, impossible to say definitely how many of these cases are in some way due to hereditary syphilis; but, considering the frequency of the association of the two, we may well keep in mind the interesting study of Hirschl,⁹ who, in the attempt to prove syphilis

Progressive Paralyse des Jugendalters. Russisches Archiv für Psychiatrie, etc., Vol. XXVI., No. 1, p. 1-130, reviewed in Centralblatt für Nervenheilkunde und Psychiatrie, XVIII., 1895, p. 610.

⁹Die Aetiologie der progressiven Paralyse. Jahrbücher für Psych., 14 Vol. iii., 1896, p. 321-541.

the cause of general paralysis, has, among other evidences, cited the results of an inquiry into a collection of cases with well-known tertiary lesions from Lang's clinic in Vienna, and has shown that there also, in spite of a most careful anamnesis, it was impossible to find a syphilitic history in all cases; indeed, taking everything into consideration, there was on this point a remarkable resemblance between general paralysis and tertiary syphilis. We must concede therefore, that even the possibility of all these cases being due to hereditary syphilis must be admitted, although this is not by any means proved, and surely never will be proved by any statistics. That our patients have such a heredity we cannot be certain, but we have seen that the father has had symptoms which were very suggestive.

There is one other point which it seems to me worth speaking of in this connection, and that is the differentiation of patients like ours from those which Homén described* as having a disease *sui generis*, most probably on the basis of a *lues hereditaria tarda*. The picture of our first patient reminded me at once very strongly of the clinical picture which Homén gives of his cases. We have here as there a disease occurring in more than one member of the same family, and here as there a progressive dementia; here as there, at least in one of our patients, contractures (although I have not seen the patient myself there can be no question about these, since I have received the same information from several members of the family independently). Homén also feels the necessity of distinguishing his cases from those of general paralysis, and the grounds on which he does it are the following: 1, the age; 2, the absence of any changes in the temper or disposition ("Laune"); 3, the absence of any delusions, and, 4, the absence of the characteristic speech disturbances. The first and third of these reasons at once prove valueless in view of our cases; nor can I see that the second, namely, the absence of changes in disposition, proves anything, not only because this certainly played an insignificant rôle in our cases, too, but because in the cases of demented

*Eine eigenthümliche, bei drei Geschwistern auftretende, typische Krankheit under der Form einer progressiven Demenz in Verbindung mit ausgedehnten Gefässveränderungen (wohl *lues hereditaria tarda*.) Arch. für Psych., Bd. xxiv., 1892, p. 191.

forms of general paralysis this feature is less frequently seen than in the more classical pictures. The strongest point certainly is the absence of the characteristic speech-disturbances; a priori we should, however, owing to the fact that they, after all, do not depend on the disease process as such, but on the localization of it, assume that cases of general paralysis could occur without it, and we certainly know that such do occur. A good case in point, for example, is that reported by Lührmann," also an instance of juvenile general paralysis. Here the symptom was lacking till death. We see, therefore, that for the present we have no sufficient data to separate the two diseases clinically. An entirely different question arises when we look at the pathological anatomical data. Here we find considerable differences. Homén found changes in the vessels which were very pronounced, not only in the brain, but in the other parts of the body as well; these he considers justly as the primary lesion. Moreover, it must strike us that the anatomical changes in Homén's cases are very slight in spite of the fact that the disease clinically was very profound and lasted for seven years in one instance; both the changes in the nerve cells and fibres and in the neuroglia were but slight. This also is, to my mind, a feature of great importance and one which points very strongly to a process which is essentially different from that of general paralysis. In other words, Homén's cases cannot be differentiated clinically from some cases of juvenile general paralysis, but anatomically they are essentially different.

SUMMARY.

It is evident that the two sisters who were in apparently good health previous to the onset of their disease, gradually developed the symptoms of general paralysis, the one beginning at the age of ten, the other at the age of fifteen years. There is a neuropathic heredity, inasmuch as the father was prematurely senile, the mother very nervous. Besides, it is probable that the father has had lues and we have seen that a luetic heredity is very common in such cases.

²²Progressive Paralyse im jugendlichen Alter. Neurol. Centralblatt, 1895, p. 632.

The clinical course in both sisters was that of a simple progressive dementia, lasting in one case for six years, in the other so far for four years. We see from the literature that this simple demented form is the most common in these juvenile cases.

The autopsy in the younger sister proved the diagnosis. The lesions in the cerebral cortex were those commonly seen in general paralysis. Similar changes were found in the basal ganglia. In the cerebellar cortex was seen a more or less pronounced degeneration of many Purkinje cells with great increase in the neuroglia of the cortex and marked diminution in the thickness of the cortex. In the cord the anterior and lateral pyramids were affected. This degeneration could be followed into the pons, but was apparently lost in the crura. From the upper dorsal cord upward there was a degeneration in Gowers' tract, the field of degeneration becoming best marked in the high cervical region and the beginning of the medulla oblongata. This degeneration is probably due to changes in the gray matter of the cord which we find in the upper portions in the centre of the gray matter on either side. We have, finally, concluded that while we have every reason to regard the cases which Homén described as differing in their essential disease process from general paralysis, it may be impossible to differentiate the two diseases clinically.

A STUDY IN APHASIA.¹

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THE study of aphasia has been developed along two distinctly diverging lines. German authors, and chief among them Wernicke and Lichtheim, have built up what we might call diagrammatic theories of speech, neglecting largely the relation that speech bears to the other faculties of mind. English writers, such as Jackson and Bastian, have dwelt upon the psychology of language and have troubled themselves far less with the relegation of speech to actual and definite cortical areas and the tracts connecting them. During the last few years the writings of various authors, particularly of Freud, Sachs, Wyllie and Dejerine, have shown the necessity of harmonizing and uniting the conclusions of the two principal methods of research employed in the study of language.

In his text-book on the Nervous Diseases of Children, Dr. Sachs, in speaking of aphasia, makes the following statement:

"Although the diagrammatic representation of language as perfected by Wernicke and Lichtheim has done much toward an understanding of aphasia, the close adherence to diagrams has prevented an appreciation of the psychic processes of language. The psychological point of view as developed by Jackson and Bastian and recently adopted by Freud, will, in connection with Wernicke's localization theories, lead to a better knowledge of the subject." In concluding his critical remarks the author expresses the belief "that in further studies on aphasia the strictly anatomical and the psychological theories must be blended with one another or must, at least, be allowed to supplement each other."

¹Read before the New York Neurological Society, Oct. 6, 1896.

A correct interpretation of the anatomical findings can, indeed, only be expected if the case was understood from a psychological point of view. It is interesting to note in this respect that cases which by some authors (Broadbent, Mills, etc.) had been explained by lesions of a special "naming-centre," have been found to belong to the group of optic aphasia, being due to an interruption of the association tracts, existing between the object representations and the auditory-speech images. A clinical observation, unsupported by an autopsy, may, in fact, occasionally be of more value than a post-mortem examination with a history incomplete from a purely clinical or psychological point of view.

In reporting the following case of (cortical) motor aphasia, with the kind permission of Dr. Sachs, I have been guided by the above considerations; I have endeavored to study the conditions as thoroughly as possible, to discover the mutual dependence of the symptoms upon each other and to give each of them its correct interpretation. This can only be done by discussing the mental processes underlying the acquisition and full development of the faculties of speech, reading and writing, and by demonstrating the intimate connection and manner of co-operation of the various cortical apparatus which the nature of these processes necessitates.

History.—Mrs. S. D., aged 23, born in the United States, of Irish parentage. Three years ago had acute articular rheumatism and during that attack probably ac-

*Dr. Sachs has kindly supplied me with much of the literature upon the subject. His valuable criticisms in reading over the manuscript of this paper, have induced me to modify some of my views, partly also to change the form of the paper. I take occasion here to express my gratitude for his kindness.

The paper was practically finished before I had read Dr. Wyllie's treatise on the disorders of speech, Dr. Elder's article (Notes on Aphasia with Special Reference to a Subdivision of Broca's Convolution) and the classical researches made in Dr. Dejerine's Clinic and Laboratory. My conclusions, particularly regarding the "internal language" and the important part which the motor-speech centre plays in the functions of reading and writing, were therefore reached quite independently of these authors, being based partly upon general psychological argumentation and partly upon a careful study of the case in question. It was a great satisfaction to find that some of my deductions were strongly supported by the ample clinical evidence which Dejerine and his pupils have brought forward, and that my views coincided in many points with those of Wyllie and Elder whose nomenclature I partly adopted when revising this paper.

quired her present heart trouble, an insufficiency and stenosis of the mitral valve. No other attacks of rheumatism since that time. Married two and a half years ago. During the first year of married life suffered an abortion in the third month of pregnancy. June 7, 1895, delivered of a child at full term. A week later had an apoplectic attack due evidently to embolism, the etiology of which must be sought for in the disease of the mitral valve. When the general effects of the lesion had passed off, the patient presented hemiplegia of the right side, with involvement chiefly of the right arm, especially of the hand and fingers. There was further complete motor aphasia and alexia, probably also complete agraphia, although on this point the patient can give no definite information. The patient claims that the understanding of spoken language was fully unimpaired; but this statement must be taken with due reserve. About two and a half months after onset, began to regain speech a little; the first words she could say were: "Yes, I want to go home." Then the speech became more fluent until she spoke as she does now (April 1896). Only about a month ago, that is, in March, 1896, she began to be able to read some.

The patient was first seen on April 3, 1896, and was examined several times during the said month. At that time the following condition was found to be present:

Rather anaemic woman. Examination of the heart reveals marked insufficiency and stenosis of the mitral valve. The patient presents a hemiplegia of the right side.

Right upper extremity paralyzed in a high degree. Extreme contractions of fingers and in elbow. No motion of fingers except very slight movement of little finger; no flexion of wrist possible and extension just noticeable. No pronation or supination to speak of. Flexion of elbow to a limited extent and inability to fully extend the forearm at the elbow. Abduction of arm and other movements in shoulder joint very limited.

Sense of pain slightly diminished in right arm; sense of touch normal.

Lower extremities.—In walking drags the right leg, which also shows distinct spasticity. Right knee-jerk exaggerated, left normal. No sensory disturbances in the lower extremities.

Functions of the auditory nerves and ocular nerves and muscles normal. Pupils normal.

Facial innervation in repose and on making voluntary movements is normal, but when smiling, the right nasal fold is less marked and the right corner of the mouth hangs a little lower than the left.

Vision. $\frac{1}{8}$ in both eyes. No narrowing of visual fields. No hemianopsia.

Speech, Reading and Writing.—The patient shows only slight disturbance of motor speech, consisting in some slight difficulty of finding the words, especially when speaking for some length of time, and manifesting itself further by occasional misuse in the proper construction of sentences.

Occasionally a question has to be repeated before she understands its meaning, otherwise the understanding of spoken language seems unimpaired. There is, however, some difficulty of naming objects presented. It is true that most of these are promptly named, but it proves that, for instance, she does not know the designation of a portrait, nor of a photograph, nor of an oil painting, as all three kinds of objects are called "pictures," and the patient is absolutely unable to find the individual names, "photograph, portrait, painting," although she must certainly have been familiar with at least the first two of these before the apopleptic seizure. It is even doubtful whether she actually recognizes the essential difference between these various kinds of "picture." Aside from these disturbances there was marked defect of the faculty of reading and writing. The patient recognizes many of the individual letters of the alphabet with great difficulty, while some are at first not recognized at all, or are mistaken for other letters. The reading of whole words is also impaired, but not proportionately to the faculty of reading the letters composing them. It is found that the patient reads many a word fluently and yet frequently spells it with great difficulty or wrongly. Occasionally she omits letters. On the whole, it is easier for her to read a word in toto than to read the letters composing it. Sometimes she reads a word wrongly and then, although looking at it, spells it in accordance with the mistake made; for instance, the word "the" is read two and spelled t, w, o. It can be at times distinctly proven, that the patient does not spell the words by looking at each single letter, but from the sound of the word after she has read the latter. This is shown from the fact that she reads "one" correctly, but spells it "w, o, n," which

has the same sound. Another example is "unknown" which she reads correctly, but spells u, n, w, n, o, w, n,.

Written text is read worse than printed, a fact which needs no explanation, since this is also the case under normal conditions

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The above reproductions of the patient's handwriting illustrate her peculiarity of using printed signs instead of script in writing.

In writing, which is done with the left hand, the patient uses printed signs instead of written ones, both in copying and in writing to dictation. Copying is done slowly and nearly mechanically. Writing to dictation causes much more difficulty than reading; the patient writes much slower and makes many more mistakes than she does in reading; sometimes she begins to write a word

correctly, but fails to finish it or completes it incorrectly. One has the distinct impression in such a case, that she has forgotten the word before she has finished writing it.

The understanding of read text seems to be entirely proportionate to her faculty of reading; that is, as soon as she has read all words composing a not too long sentence, she as a rule readily grasps the meaning of this sentence; but if the reading of individual words of a sentence causes much strain, the difficulty of deciphering the words make her lose connection between them; she has forgotten the first words while reading the last. There is no doubt, that the auditory impressions received from loud reading help the understanding and enable her to decipher words a posteriori, which she had not been able to decipher before. That is she sometimes fails to read a word or misreads it, but recognizes her mistake as soon as she has read the words following it.

In some respects the examination had to remain incomplete, as the patient was lost sight of. I may emphasize this however, that she had absolutely no visual disturbances. Vision $\frac{10}{10}$ in both eyes. No hemianopsia. No narrowing of the visual fields. Entirely normal perception of colors.

The memory was not carefully tested. The intention to do so was frustrated by the non-appearance of the patient; but the fact alone that she made a rapid progress in the faculty of reading during the sessions devoted to the test of this faculty, indicates the absence of gross lesions of memory. It seems appropriate here also to make some remarks on the patient's mental attainments. The patient had been a clerk in a dry goods store. Her duty was to show goods to the customers and to write out orders for the goods bought. This tends to show that she had considerable practice in writing, as in the rush of business, the writing had to be done rather hurriedly. Aside from such routine writing she did some correspondence, writing about one or two letters a week to friends. On the whole, however, she did much more reading than writing, and read for the most part from printed text, chiefly from novels and the like. She had acquired the faculty of reading, by means of the old method of learning first the single letters and then the reading of words by spelling. Her education corresponded to that of a girl passing through the upper grades of a grammar school.

Before entering upon a discussion of the symptoms in the case reported I wish to refer to some recent investigations which may help to enlighten us on the nature and function of that part of the brain which heretofore has been designated as the motor-speech centre. I shall first mention the researches of Bianchi,³ which, although not bearing directly upon this question, may yet serve to throw some light on it in an indirect way.

Bianchi has recently studied the effects of the removal of the frontal lobes in monkeys and has arrived at the following conclusions regarding the functions of this part of the brain:

The frontal lobes preside over certain higher mental faculties and are, so to say, a centre for the intelligence. They constitute the organ in which the various sensory and motor images deposited in the different centres of the cortex become coordinated and fused. The destruction of this region entails the loss of the anatomical and physiological basis upon which judgment and the reasoning faculties are reared. These defects were very evident in monkeys deprived of both frontal lobes.

The anatomical results greatly harmonize with the conclusions to which the author had been led by study of the mental condition of such animals. Bianchi found hardly any degenerated fibres in the internal capsule of a monkey whose frontal lobes had been removed and who was allowed to live eleven months after the operation. He concludes from these observations that the frontal lobe contains but a very small number of projection fibres. He found, however, extensive degeneration in other regions, showing that the frontal lobe possesses a vast associative corona radiata, the fibres of which distribute themselves to the motor district and to the sensory areas. The association with the occipital lobe is established chiefly by means of the fasciculus occipito-frontalis, the connection with the temporal lobe principally by means of the capsula externa, possibly, also, of the capsula extrema and of the fasciculus uncinatus, while the association with the central convolutions occurs partly by means of the fasciculus arcuatus.

So far Bianchi. It remains to show in how much the observations of this author can contribute to the elucidation

³Bianchi: *Annali di Nevrologia*, 1895, p. 149.

tion of the physiology and psychology of articulate speech. The experiments upon animals cannot, of course, give us any information on the function and position of the speech centres. But in accepting Bianchi's theory the following question should be answered: Shall we consider Broca's centre as part of the frontal lobe in the sense of Bianchi's definition of this organ or shall we count it among the motor areas of the cortex?

Until recently Broca's centre has commonly been considered as motor. One has spoken of motor word images or motor memories of speech, in contrast to auditory word memories. The anatomical position of this so-called "motor-speech centre" would, indeed, not contradict such a view, since it is directly contiguous with that area of the cortex which we are accustomed to call the motor district and might, therefore, be considered as a part of it. But, on the other hand, the function of Broca's centre is so much more complex than that of the parts composing the motor district proper that we hesitate to put it on one line with these. From the result of recent observations we further become more and more convinced that, while the cortex of the motor district is directly connected with the gray masses of the cerebral axis, the connection of the so-called motor-speech centre with these gray masses is indirect, being interrupted by cells of other regions of the cortex. It seems to be very probable that the fibres emanating from Broca's centre find their ending place in that division of the cortex where the centres for the larynx, pharynx, tongue and the lower part of the face are situated, and that a second neuron is needed to establish the connection with the nuclei of the pons and medulla. If such is the case we must conclude that Broca's centre is a centre of a higher order, presiding over the centres of the larynx, pharynx, tongue and the lower part of the face. It will then remain to demonstrate that it not only presides over the motor centres mentioned, but has at the same time the function of fusion of sensory and motor elements, and must, consequently, be considered as a part of the frontal lobe in the sense of Bianchi's definition.

In advancing such a hypothesis I wish to point out that it greatly harmonizes with the view which Wyllie⁴ expresses on this subject. In his excellent book on the dis-

⁴Wyllie: *The Disorders of Speech*. Edinburgh, 1894.

orders of speech we meet the following statement: "It seems to me that the foot of the third frontal convolution is not motor in function and yet has relations of extreme importance to the motor production of speech; we are almost forced to the conclusion that it must be in a special manner the storehouse of the psychological pictures or memories which in themselves are so largely sensory in constitution, being made up chiefly from memories of muscular and tactile sensations."

The assertion that the motor-speech centre is a centre of a higher order presiding over the centres of the larynx, pharynx, etc., finds further support in Elder's⁵ observation. This author had the opportunity to perform the autopsy on a case with the following history [given in abstract]:

In a man of sixty, always well and strong before, speech suddenly became affected. The next day one could notice paresis of the right side of the face, not involving the orbicularis palpebrarum, however. He spoke in a thick and very blurred manner so that it was difficult for him to pronounce the words distinctly. This, however, was seen to be due simply to a difficulty he had in moving the tongue, lips and other muscles of articulation as readily as he wished to. There was no real aphasia, he knew what he was going to say, tried to say it and always succeeded in saying it, but the words were blurred and indistinct. Difficulty in swallowing. The voice unimpaired in every respect. No paralysis of the arms or legs. Five days afterwards he grew weaker, showed signs of hypostatic congestion in the base of the lungs, and died the next day.

The autopsy report of this case reads as follows:

On slicing the brain horizontally a blood clot of about a dessertspoonful in quantity was found at the level of the lower part of the ascending frontal and ascending parietal convolutions of the left hemisphere (see Fig. 1). It had destroyed almost entirely the cortical substance of the lower end of these convolutions, from the Sylvian fissure as high up as the level of the fissure that divides the second from the third frontal convolution. It left intact the third frontal convolution and that region of the lower part of the ascending frontal which adjoins the precentral sulcus.

⁵ Elder: Edinburgh Hospital Reports, 1895, p. 463.

The findings of the case induce Elder to substantiate Wyllie's view and to subdivide Broca's convolution [in the old sense of the term] in the following manner (see Fig. 2):

- (a) Centre of the psycho-motor images of speech, occupying the foot of the third frontal convolution between the precentral sulcus and the anterior ascending branch of the Sylvian fissure.
- (b) Centre for the adduction of the vocal cord or phonation centre—which was intact in Elder's case—occupying the anterior part of the lower end of the ascending frontal convolution, that is, the area adjoining the centre of the "psycho-motor images."
- (c and d) The articulo-motor centre—nearly entirely destroyed in Elder's case—occupying the lower part of the ascending parietal convolutions with the exception of the area occupied by the centre (phonation).
- (g) Probably there is also a respiratory centre near by, the action of which is required in speaking.

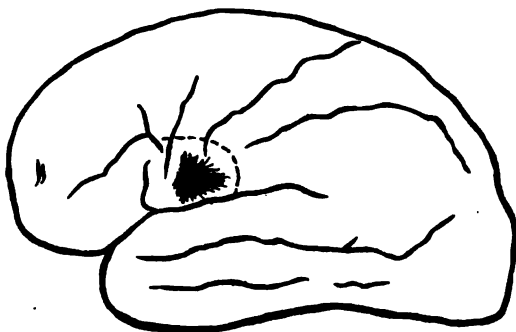


Fig. 1.—Position of focus in a case which had presented dysarthritic but no aphasic symptoms (Elder).

In accepting this subdivision and in assuming that the centre of the psycho-motor images is associated with the three (or two?) sub-centres mentioned, the creation of the type of subcortical motor aphasia (Wernicke) or aphasia motrice pure (Dejerine) finds its justification and would be produced by an interruption of the said association tract, which presumably is contained within the fasciculus

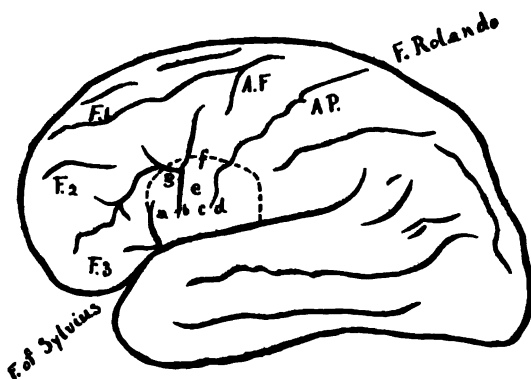


Fig. II.—Subdivision of Broca's convolution after Elder.

- a. Psycho-motor speech area or centre of the psycho-motor images of speech (Wyllie).
- b. Centre for the adduction of the vocal cords (phonation centre).
- c. and d. articulo-motor centre (centre for the muscles of the tongue, throat and opening and closing of mouth—Horsley and Beevor).
- e. Centre for the lower face and angle of mouth (Horsley and Beevor).
- f. Centre for upper part of face (Horsley and Beevor).
- g. Centre for acceleration of respiratory movements of the larynx.

arcuatus. H. Sachs¹ also inclines towards this view. Dejerine² finds subcortical motor aphasia due to destruction of the projection bundles of the third frontal convolution, which practically means the same thing, as by such destruction the connection of the centre of the "psycho-motor" images of speech with the sub-centres of Broca's area must also become interrupted.

With the acceptance of Wernicke's old theory of the existence of a special "speech tract" (Sprach-bahn) connecting the motor-speech centre [that is, the centre of the psycho-motor images] directly with the nuclei of the medulla, a pure type of subcortical motor aphasia could not be conceived, as it could not be imagined without the co-existence of anarthritic or dysarthritic disturbances. Wernicke³ has finally given up this theory, and it is only strange that he clung to it so long.

Whenever in the further course of this paper the terms

¹H. Sachs: Vorträge über den Bau und die Thätigkeit des Grosshirns und die Lehre von der Aphasie und Seelenblindheit. Edition of 1893.

²Quoted from Mirallié: L'aphasie sensorielle, p. 101.

³According to a quotation of H. Sachs, l. c.

"motor-speech centre" or "Broca's centre" or "Broca's area," are used, it is meant to designate by these terms only that part of Broca's convolution which Wyllie and Elder call the centre of the psychomotor images of speech. This distinction will be desirable, as only lesions of the centre of the psychomotor images should produce lesions of the mental or "internal language"—and, accordingly, alexia and agraphia, as will be shown later on—while lesions of Elder's "subcentres of Broca's convolution" should cause only the impossibility or disturbance of articulate or external speech, leaving the so-called internal or mental language intact and, consequently, not causing any disorders of the faculties of writing and of mental reading.

(To be continued.)

Clinical Cases.

ACUTE ANTERIOR POLIOMYELITIS, ASSOCIATED WITH FACIAL PALSY.¹

By LEO STIEGLITZ, M.D.

Neurologist to the Mount Sinai Hospital Dispensary; Assistant in Neurology, Vanderbilt Clinic.

THIS little patient, Frank G., six years of age, was brought to Dr. Starr's Clinic for Nervous Diseases at the Vanderbilt clinic² on Oct. 13, with the following history:

The boy had always been a healthy child till the 28th of September, on which day the mother noticed that the right side of his face was paralyzed; she is positive that the boy was otherwise perfectly well at the time and had no fever. Five days later the child was taken at night with a fit of vomiting and high fever; the fever lasted about three days, the boy seeming quite ill during its duration. The evening of the second day of this illness Mrs. G. noticed that the child had lost the power of its left arm; two days later the right arm became also powerless. During this time the boy complained very much of pains in all his limbs and cried when they were handled, for instance, when an attempt was made to rub them with alcohol. The mother noticed no weakness in the legs till the boy began to go about again, when he had difficulty in mounting stairs and wanted to be carried. At no time was there any trouble in micturition or defecation.

Immediately before his illness the boy had been apparently in perfect health, there was especially not the slightest indication of any sore throat. The child was never given any liquor and careful investigation failed to reveal any source of any metallic poisoning. Three months previous to this boy's illness Mrs. G. had lost a boy three years of age from tubercular meningitis, at least that was the

¹Read before the New York Neurological Society, Nov. 3, 1896.

²I am indebted to Dr. Starr for the privilege of presenting the case to the Society.

diagnosis made by the attending physician. The mother states positively that the child's condition did not resemble that of our little patient. A brother of Mrs. G. suffered a paraplegia of the legs in infancy, recovered the use of one leg and remained paralyzed in the other leg for the rest of his life, a condition, I believe, we can safely regard as due to an acute infantile spinal paralysis.

Examination.—The boy is very anaemic, the adipose tissue poorly developed. Temperature at all examinations normal.

Face.—The pupils are equal, respond well to light and accommodation. There is no difficulty in swallowing, no disturbance of sight or hearing. There is no ophthalmoplegia. There is complete facial palsy of the right side; very slight contractions are possible in the corrugator supercilii. The left side of the face is unimpaired, the tongue is protruded perfectly straight, the soft palate contracts equally well on both sides. The electrical examination shows greatly diminished excitability of the right facial nerve to both currents; a very strong, painful faradic current is required to induce by direct stimulation a slight contraction in the muscles of the mouth, a somewhat weaker current is required with the orbicularis oculi. The galvanic formula obtained from the lower lip reads 0, 8 MA A C C; 1, 5 MA K C C, from the orbicularis oculi 1, 5 MA A C C; 2, 0 MA K C C; the contractions are sluggish in character. In other words, we have on the right side of the face partial R D tending towards complete R D. The sense of taste is unimpaired on both sides.

Upper Extremities.—Right arm: There is complete paralysis of the deltoid, biceps, brachialis internus and long supinator muscles and almost complete palsy of the triceps. The electrical examination shows complete R D in the muscles completely paralyzed, in the triceps the faradic excitability is diminished and A C C=K C C. The other muscles of the right arm are intact, both as regards motor power and the electrical excitability.

Left Arm: There is complete palsy of both flexors of the wrist and of the thenar muscles. There is a very marked loss of power in the flexors of the fingers, in the extensors of the wrist and fingers and in the interossei muscles. There is some weakness of the biceps. The deltoid, the flexors of the elbow and the long supinator show no loss of power, and their responses to the electri-

cal currents are normal. The triceps shows diminished excitability to the faradic current, and $A C C > K C C$. The long extensors of the wrist and fingers show diminished faradic excitability, normal galvanic responses. The flexors respond to a very strong faradic current with a sluggish contraction, their galvanic excitability is increased, $A C C > K C C$. Of the muscles composing the thenar the flexor pollicis brevis shows but slight changes in the electrical excitability, whereas the abductor pollicis brevis does not respond at all to faradism and presents the galvanic formula $A C C$ 0.75 MA, $K C C$ 1.5 MA, the contractions are very sluggish, barely perceptible. There is marked atrophy of the thenar. The abductor Digit IV. and the Mm. interossei, with the exception of the M. interosseus III., show loss of faradic excitability; they respond readily, but with a sluggish contraction, to the galvanic current, $A C C$ predominating over $K C C$. The M. interosseus III. responds to the faradic current well, but shows $A C C$ 0.3 MA, $C C C$ 1.0 MA. The electrical examination of the three large nerve trunks shows normal conditions on the right side. On the left a fairly strong faradic current applied to the ulnar nerve will induce contractions in the long flexors of the fingers supplied by that nerve; the very strongest current fails to elicit any response from the ulnar flexor of the wrist. The median and musculo-spiral nerves of the left arm show diminished faradic and galvanic excitability.

Legs.—The left quadriceps femoris is weaker than the right. In mounting stairs the boy uses only his right leg to raise himself from one step to the next; when told to try with his left leg he places the foot on the step above but is utterly unable to raise his body by straightening out the leg. Before his illness he mounted stairs in the usual way, using his legs alternately. None of the other muscles of either leg shows the least loss of power. The right knee-jerk is lively, the left is diminished. Repeated examinations failed to reveal any disturbance of sensation in any part of the body, nor have there been any paraesthesia present at any time. The nerve-trunks of both arms were found to be slightly sensitive to pressure at the first examination, but the boy's statements in this respect have been changeable and unreliable. The paralyzed muscles have been certainly sensitive to pressure.

The further observation of the case has shown a grad-

ual recovery of motor power in a number of the affected muscles, most noticeably in the triceps of the right arm, the flexors of the fingers, the extensors of the wrist and fingers and the interossei of the left arm and the left quadriceps; two or three days ago the boy commenced mounting stairs again with his left leg.

To recapitulate briefly the conditions presented by this case, we have a palsy of the right side of the face, followed five days later by an acute febrile attack of two or three days' duration; during the febrile condition an asymmetrical paralysis of both arms, with slight involvement of one leg, associated with pains in all four extremities developed.

In arriving at a diagnosis in this unusual case of infantile paralysis we have the following three conditions to choose from: (1) An acute infectious multiple neuritis, with involvement of the facial nerve; (2) an acute anterior poliomyelitis with involvement of the right facial nucleus, (3) an acute anterior poliomyelitis complicated by a peripheral facial neuritis of the ordinary type (Bell's palsy).

There are three points which favor the diagnosis of a multiple neuritis; the first is the presence of facial palsy. Facial paralysis in acute poliomyelitis is very rare indeed, a great deal rarer than in multiple neuritis; Oppenheim, for instance, states that in a doubtful case the involvement of cerebral nerves would point strongly to multiple neuritis.* The persistency of the pains in the limbs beyond the first stage of the disease is a second point, and a third one is the apparent tenderness of the nerves of the arms to pressure. This symptom influenced the diagnosis originally made to a considerable extent, but I was not able to verify it at any subsequent examination. An acute febrile onset of multiple neuritis is occasionally observed, sometimes in epidemic form even in our climates (Eisenlohr,[†] Bordurant,[‡] Medin[§]). It is not impossible in fact that the same infectious agent which is active in acute poliomyelitis will sometimes produce an acute infectious form of multiple neuritis; Medin, for instance, observed cases of this kind in the midst of the epi-

*Oppenheim: *Nerven-Krankheiten*, 1894, pp. 155 and 352.

†*Berliner klinische Wochenschrift*.

‡*Medical News*, Oct. 3, 1896.

§*Transactions of the X International Congress*, Vol. II.

dem of poliomyelitis which he described before the Tenth International Congress in Berlin. Nor did the absence of objective sensory disturbances such as are usually present in multiple neuritis make the diagnosis of this disease in our case untenable, as undoubted cases of the kind are seen in which a most thorough examination fails to reveal any impairment of sensibility. These were the considerations which caused me after the first examination to look upon the case as one of multiple neuritis. A more careful analysis, however, led to an entirely different conception. In the first place, and this is a very important point to my mind, the distribution of the paralysis presented by the patient does not accord at all with the symmetry considered characteristic of multiple neuritis; furthermore, the latter disease injures, as a rule, the distal groups of muscles, those of the forearm and of the lower leg to a greater degree than the muscles controlling the movements of the elbow and knee. In our case the greatest paralysis is present in the right upper arm and in the legs the only muscle affected is the quadriceps femoris. The reverse of all this is true of acute poliomyelitis; the distribution of the paralysis in our case is almost pathognomonic of infantile spinal paralysis, a fact which first aroused my doubts as to the correctness of the original diagnosis. The paralysis of the right arm corresponds to the upper arm type of Remak, the involvement of the long supinator being especially characteristic. In the left arm, on the other hand, we have a very marked loss of power in the extensors of the wrist and fingers and perfect freedom of the long supinator supplied by the same nerve. In fact, with the aid of Starr's¹ and Thorburn's² tables we can readily map out the affected segments in the cord; a lesion in the right half of the V. cervical segment suffices to account for the permanent paralysis in the right arm, a lesion located in the VIII. cervical and I. dorsal segments, extending upward into the VII. cervical fully explains the condition in the left arm. The isolated paralysis of the quadriceps femoris is met with so frequently in infantile spinal paralysis that it furnishes a strong support of this view of the case. I need hardly state that the acute onset of the paralysis, with fever and vomiting, is the rule in poliomyelitis as it is the exception in multiple neuritis.

¹Starr: American Journal of Neurology and Psychiatry, Aug. 1, 1884.

²Thorburn: Diseases and Injuries of the Spinal Cord.

The pains the boy complained of are frequently observed during the febrile stage of poliomyelitis and are easily accounted for by the collateral congestion of the posterior gray matter, and, especially, by the tract recently described by Ciaglinski⁹, nor is there any valid reason why these pains should not persist in a given case longer than in the general run of cases.¹⁰ Thus, I believe, we would have no difficulty in classifying this case as one of poliomyelitis but for the presence of one feature—the facial palsy. For the involvement of cerebral nerves in infantile spinal paralysis, though not entirely unknown, is so rare a condition that a case of the kind must be sifted thoroughly before it can be allowed to pass muster as such. Gowers¹¹ states that he has seen but a single instance of the kind and quotes a second characteristic case, recorded by W. Pasteur.¹² Medin,¹³ however, reports no less than seven cases observed during the epidemic in Sweden in which cerebral nerve palsy accompanied poliomyelitis; in two fatal cases Rissler¹⁴ demonstrated microscopically changes in the nuclei of the hypoglossus and vagus. Caverly¹⁵ reports one case with facial palsy in his account of the epidemic in Vermont two years ago. With these various cases as precedents we might feel justified in regarding ours as a further case of acute poliomyelitis with bulbar involvement. Nevertheless, I am not prepared to accept such a view. Let us recall the course of events in our case. Without the slightest disturbance of the general condition, as the mother repeatedly assured me, facial palsy developed; five days later, during which interval the child was otherwise apparently perfectly well, high fever and vomiting suddenly set in, and two or three days later the paralysis of the limbs developed. Gowers does not state whether the facial palsy developed in his case simultaneously with the rest of the palsy. In all of Medin's cases the fever and general symp-

⁹Neurol. Centralblatt, No. 17, 1896, p. 773.

¹⁰A further point, worthy of mention as militating against the diagnosis of multiple neuritis, is the fact, that if the face is affected in this disease there is always *bilateral* palsy (Gowers, p. 235, Vol. II.)

¹¹Gowers, Diseases of the Nervous System, Vol. I., p. 363; Vol. II. pp. 231 and 240.

¹²Lancet, 1887, II., p. 858.

¹³Loc. cit.

¹⁴See Medin's article.

¹⁵Med. Record, 1894, Dec. 1. Case 88.

toms preceded the facial palsy, which developed either at the same time or within one or two days of the rest of the paralysis. It is true, he saw in a few cases fever and vomiting, followed by slight paralysis, then a non-febrile interval of five or six days and, finally, a recurrence of the fever, generally in increased severity, and paralysis of new groups of muscles. But in no case did paralysis precede the systemic disturbance; in three cases of facial monoplegia occurring during the epidemic he is careful to state that the paralysis was preceded by the characteristic febrile attacks. In Caverly's case the facial palsy is said to have occurred simultaneously with the fever and nausea, the general paralysis following two days later. The point I wish to emphasize is the fact that in none of these cases did the facial palsy precede the onset of fever, as little as we see paralysis of an arm or leg develop before the fever. Such an occurrence would clash with our whole conception of acute poliomyelitis as an acute infectious disease, in which the systemic symptoms of the infection precede the symptoms referable to the localized lesions very much as the fever and vomiting of scarlatina precede the rash.

These considerations lead me to regard our case as one of acute poliomyelitis associated with a coincident Bell's palsy of the ordinary type. It need hardly be added in closing that an exact diagnosis in a case of this kind is of practical importance as well as of scientific interest, inasmuch as the prognosis quoad restitutionem ad integrum of the paralysed muscles differs materially in the two diseases which came into question. In the case before us, for instance, the boy will surely retain a permanent loss of power in the paralyzed muscles of the right upper arm and, probably, will not recover the use of the flexor carpi ulnaris and of the thenar muscles in the left arm; whereas, if it were a case of multiple neuritis even the complete R D in these muscles would not justify an unfavorable view as to the ultimate return of power, though it might require a long time to set in.

Note—Jan. 23, 1897.—The further observation of the case has confirmed the view taken above. The facial palsy is running the course of an ordinary Bell's palsy of medium severity, and most of the affected muscles of the left arm have improved considerably. The deltoid, biceps, brachialis internus and long supinator of the right arm have wasted away and show absolutely no sign of any return of power.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting Oct. 6, 1896. B. Sachs, M. D., President.

A CONTRIBUTION TO THE STUDY OF MOTOR APHASIA.

Dr. B. Onuf read a paper with this title. (See this journal, page 86).

DISCUSSION.

Dr. Joseph Collins said that he agreed for the most part with the views expressed by the reader of the paper. Certainly the paper served to still further confirm the views expressed by Bianchi. Personally, it seemed to him that all that a study of aphasia could do was to help the pedologist. We could do a great deal for psychology by working in the field of aphasic disturbances. He could not agree with Dr. Onuf, that a person having pure motor aphasia had complete inability to read. It did not seem to him that it was necessary in order to read to translate what we read into articulate words, and he thought he had one or two cases under observation at present which would abundantly substantiate this statement. One patient was completely aphasic, yet he could write prolifically and was able to read and understood those writings; hence, the speaker said, he could not believe that with pure and complete motor aphasia there is alexia. He did not think there was any objection to subdividing the motor speech centre into an articulo-motor centre and placing adjacent to it the centres for phonation, labial movements and buccal movements.

The President said that his individual experience with aphasia had been a rather curious one. At first, he had been impressed with the writings of Jackson and Bastian. Then had come the German school with all sorts of mechanical theories, and now we all felt the need for returning to a mixture of the psychological and the mechanical theories. On the whole, he was in entire agreement with the views expressed by the reader of the paper. Recently he had observed the progress of a case in which aphasia was the sole symptom of a

cortical tumor. The manner in which it progressed would seem to justify fully the subdivision of the motor speech centre, and also to show that speech is not the function of any one centre, or any series of centres, but that it is really the result of a very close union of those centres by distinct association tracts. If this were not so a relatively small lesion could not explain the variety of symptoms observed in a single case. The case referred to was that of a lady who had been carefully observed by her brother, who was a physician. The first thing noticed was an apparently slight apathy, but this was really due to a difficulty in speech. When first seen by Dr. Sachs, about three months after this, it was found that she had lost the faculty of using nouns, so that she could not give the names of those persons best known to her. After awhile, the difficulty of speech became more distinct; there was great difficulty in finding words. Towards the end only was there a distinct deficiency in the understanding of language. From the first her brother had noticed a distinct difficulty in reading and a still greater difficulty in writing. The speaker had never seen a patient able to speak so much and yet be unable to name or even copy single letters. She could, however, write a *whole* name fairly well. Such a case seemed to show the necessity for a further subdivision of the motor area. The paper of the evening was of value as a corroboration of Bianchi's views. It showed that we were gradually turning to larger divisions rather than to the small localization areas which we employed as a result of the teachings of Ferrier.

Dr. Onuf, in closing the discussion, said that he distinguished two forms of aphasia—cortical and subcortical motor aphasia. The latter was also called pure motor aphasia. In cortical motor aphasia it was assumed that the cortex, where psycho-motor images of speech are deposited, is affected. Subcortical aphasia is one in which there is an impossibility of loud speech, but internal language remains intact; hence, such persons can read and write perfectly. He believed the cases referred to by Dr. Collins were examples of subcortical motor aphasia.

THE COMMITMENT OF PATIENTS AND THE NEW INSANITY LAW.

Dr. G. W. Jacoby read a paper on this subject. He said that under the name of the insanity law there went into effect a new law in July of this year. In his opinion, the framers of the law had totally failed to unite the postulates of jurisprudence with those of medicine; indeed, it would seem that they had intentionally ignored, as far as possible, the medical side of the subject. The medical certificate no longer serves for the temporary detention of the patient for five days. The responsibility for the commitment has been removed from the shoulders of the physicians to those of the judge, and a matter which is essentially medical has been transformed into one chiefly legal. This law also provides that at least one day before the physician presents his application to the judge the patient is to be informed of the proceedings. This personal service can be omitted under certain circumstances, according to the discretion of the judge. After all the necessary legal preliminaries have been taken, the superintendent of the institution to which the patient is committed may refuse to accept the patient on the ground that he does not consider the person insane or that the papers are not made out properly. There is also a provision for an appeal from the decision of the judge and a trial by jury. The old law was much better, particularly on account of its provision for temporary detention. The provision which takes away every method of procedure except appeal, when the application is refused, is a particularly objectionable feature. Personally, he would not be satisfied with an insanity law which would not allow of the temporary commitment of the person on the strength of medical certificates by two qualified physicians, one of whom should have special psychiatric qualifications.

Dr. Carlos F. Macdonald said that he wished at the outset to disclaim any responsibility for the framing of the law. It had its origin with a member of the Statutory Committee of Revision. He was the only physician in this State who had opposed the bill before the Legislature. During the past seven years, as Commissioner in Lunacy, he had examined thousands of cases of alleged illegal commitment, and he had yet to learn of a single case of a sane person being committed through corrupt collusion and through intent, although he had occasionally known of instances of mistaken diagnosis,

such as might occur in connection with any disease. He thought that as a rule judges would waive the notice of personal service upon the patient or friend, and it seemed to him a distinct advantage to make the commitment a judicial order rather than a judicial approval, as in this way it relieved the medical profession of much responsibility and the danger of suits for damages. In his judgment, the weakest point was the absence of any provision for temporary detention. A determined effort should be made this winter by the medical profession to amend the law in that respect. Curiously enough, the laity consider themselves fully as qualified as physicians to diagnosticate insanity and this new law is an outgrowth of that feeling.

Dr. C. L. Dana said that his views were entirely in harmony with those of the reader of the paper, and the society should make it clear that it appreciated the absurdities and many faults of the new law. It had caused an infinite amount of trouble to the city physicians and the ordinary process of commitment had, in consequence, become tedious and expensive, so much so that physicians had found it advisable to commit insane persons, as far as possible, to institutions outside of this State.

Dr. F. Peterson said that the new law was objectionable in that the paper must be fully made out and approved by the judge before the patient could be sent to an asylum, and because of the possibility of other difficulties arising in practice, such as had been mentioned. In his own experience, however, the judge had in every instance dispensed with the personal service.

Dr. Graeme M. Hammond said that he agreed with the views presented in the paper, and now that the evils had been pointed out it was our duty to consider the best remedy. He hoped the society would take an active interest, for instance, by appointing a committee charged with the duty of urging proper amendments.

Dr. L. C. Gray said that he thought the law was not only absurd but an outrage. It was ridiculous that physicians should submit to lawyers about a matter involving the question of a disease of the brain.

Dr. Collins said that according to the new law the physician certified to the insanity of the person and the judge committed him. This was no infringement upon the rights of the medical profession. He did not think that the gloomy view and the objections presented in the paper were well founded. It was true there were some objectionable features, but they did not appear to him to be of vital importance. The new law offered unusual opportunities for a trial of the home treatment of the insane, so strenuously advocated by some.

Dr. L. F. Bishop said that the new law had given him less trouble than the old law.

Dr. Hirsch said that this law had no counterpart in any other country, for not only the disposal of the lunatic, but the decision as to his insanity was made by the judge, the physicians only giving their testimony. This testimony was given on special blanks which made it far from scientific. It was remarkable that a judge must tell us whether a patient is fit to live with his family or must be deprived of his liberty because of his being insane and a menace to society. Persons having small pox or cholera were often forcibly removed from their homes, but in these instances the decision was made by a physician, as it should be, and not by a judge. He thought that the personal service provision was objectionable and sometimes produced a bad mental impression on the patient.

Dr. M. Allen Starr said that according to the new law the judge acted entirely upon the testimony given by the two physicians; the physicians' rights were more fully protected than by the old law. He urged that the society should not take an extreme view, but should simply ask that one or two features, which were generally admitted to be particularly objectionable, should be amended.

The President said that the worst feature was the lack of provision for temporarily detaining acute cases. One result of the new law would probably be the establishment of many private institutions for the care of the insane which would not be under the control of the State. He had found that it took about four hours to have a certificate signed in this city.

Dr. Jacoby, in closing the discussion, said that he did not look upon the new insanity law as an unmitigated nuisance. The judges now take the certificates of the physician simply as testimony additional to that of the petitioner. If an appeal should be taken from the decision of the judge it was not clear that adequate provision would be made for the patient.

Dr. Hammond then offered the following resolution, which was unanimously adopted:

Resolved, That the President appoint a committee of five to report to the society such measures as it may deem expedient for securing the amendment of the present lunacy law governing the commitment of the insane.

Stated Meeting November 5, 1896.

REVISION OF THE LUNACY LAW.

The President appointed the following committee: Drs. G. M. Hammond, G. W. Jacoby, M. Allen Starr, C. L. Dana and Joseph Collins.

Dr. Stieglitz presented a case of Acute Anterior Poliomyelitis, Associated with Facial Palsy. (See this number, p. 98).

Dr. M. Allen Starr said that he had examined the case very carefully at the clinic, and coincided entirely with the diagnosis made by Dr. Stieglitz, i. e., that there were present two independent affections.

Dr. Terriberry said that the boy still complained of considerable pain when the right arm was moved. One would hardly expect to find such a sensory condition at this stage if the case were one of poliomyelitis. He inclined rather to the view that the case was one of multiple neuritis. Taking this view of the case, the face palsy would coincide with the rest of the condition.

Dr. G. W. Jacoby said that the emphasis laid upon the sensory symptoms would certainly lead one to think that the neuritic symptoms preponderated; hence, he saw no reason to classify the case in either one or the other category. The case corresponded very closely with those described as combined cases of poliomyelitis and neuritis.

Dr. Starr said that in his experience pain was a common symptom of anterior poliomyelitis. In not a single febrile case seen by him in the last four years had pain been absent. We must, of course, separate the febrile from the afebrile cases. In the latter class the pain was not ordinarily present. He had now under his care a little girl who had the attack last August and still she suffered a great deal of pain even at present. In this case the distribution of the paralysis was thoroughly characteristic of anterior poliomyelitis.

Dr. Edward D. Fisher thought that the distinction should be made in the pain. The pain in the cases of neuritis was found especially along the course of the nerve, whereas the pain in the cases of poliomyelitis was more in the muscles, or upon movement of the muscles.

The President said that he had already published the statement that pain was a particularly significant symptom in the early stages of anterior poliomyelitis. Children in the early stages of this disease almost always show pain to a marked degree, and it was often very difficult in practice to make the distinction alluded to by the last speaker. He had now under his care a child whose leg was very tender, although now in the sixth week of the disease. On the other hand, he believed that the two conditions might occur in the same patient, and he could see no absolute necessity for these strict clinical classifications in every case.

Dr. Stieglitz said that the prognosis depended very much upon the exact diagnosis, and hence, these distinctions were of practical importance. The case he had presented had no paraesthesiae and the nerve trunks were no longer sensitive to pressure, although the muscles still exhibited some sensitiveness.

Dr. Philip Meirowitz presented a man, fifty-seven years of age, suffering from compression of the spinal cord by an hydatid cyst in the post-compression stage. In 1852 he had a severe attack of inflammatory rheumatism, and it was nine months before he had been entirely well. From 1882 to 1887 the extremities were the seat of pain, apparently rheumatic in character. In 1889 a small tumor developed to the right of the lower part of the spine. In 1893 a second tumor appeared in the lower part of the right dorsal region, and shortly after this a third swelling made its appearance between these two. He subsequently developed weakness in the lower extremities and difficulty in evacuating the bladder, but there had been no dribbling of urine. He experienced a sensation of coldness in the lower extremities and on the body up to the umbilicus. The sexual function had been gradually lost. On May 15, 1896, the examination showed a large swelling, consisting of three tumors, in the lower part of the back, to the right of the vertebral column. This tumor was distinctly fluctuating. The man walked at this time with extreme difficulty. Sensation was diminished in certain areas, but in no place was it entirely lost. The patellar reflexes were markedly exaggerated. There was a diminution of electrical contractility in the muscles. A clear, colorless fluid was drawn off from the spinal tumor and microscopical examination showed the hooklets of the echinococcus. Dr. Samuel Lloyd operated upon the case on June 13, 1896, with the most gratifying results. The speaker examined the man again on Oct. 11th. At that time the paraplegia had entirely disappeared and he was able to walk long dis-

tances without assistance. Tactile sensation was still slightly diminished. The functions of the bladder were much improved, as were also the sexual functions.

Dr. S. Lloyd said that the tumor had extended from the middle of the scapula down across the sacrum. A tumor the size of a foetal head dipped in toward the abdomen on the right side. A point of interest in the case was the diagnosis. As the cyst was a multilocular one, he had not been able to accept the diagnosis that had been previously made of cysto-sarcoma and, owing to the localization of the metastasis, he had been led to doubt the diagnosis of multiple sarcomata. Another possible condition was a large hydro-nephrosis, but the diagnosis had been settled by microscopical examination. It was probable that the cyst had not originated in the spine, but had extended into the spine. The point of entrance of the tumor into the spine was between the laminæ of the eighth and ninth vertebræ. This exactly corresponded with the localization made by Dr. Meirowitz. One old cyst and three smaller ones were removed from the cord at that point, but no effort was made to explore further on account of the already extensive operation demanded.

THE PATHOLOGY AND TREATMENT OF MIGRAINE.

Dr. C. A. Herter read a paper with this title. The treatment to be advocated, he said, was a departure from that usually followed, although not original with him. The typical migraine paroxysm seemed to be almost always associated with nutritive disturbances, which should be considered as part of the migraine paroxysm. Too much attention, he thought, had usually been given to the relief of the pain. There were many features in a typical attack of migraine indicating gastro-intestinal disturbance. During the period of marked headache there was usually no great evidence of intestinal derangement, although there was often at least slight constipation. The fæces generally appeared normal. The urine passed during the period of marked headache was exceedingly scanty, high colored and of high specific gravity and acid. Apparently the quantity of uric acid was diminished, and the excretion of urea and the chlorides was regularly diminished, due chiefly to the temporary abstinence from food. The ingestion of food often caused severe nausea and vomiting, but sometimes it was significant that it caused a return of the hemicrania. There was a rapid transition from the period of diminished excretion to that of increased excretion

sometimes within twenty-four hours. Usually after a severe migraine paroxysm the urine was increased in amount on the second and third days after the headache. The uric acid was increased and the extractives also. In seven different patients he had had an opportunity of examining the contents of the stomach in the paroxysm. In these cases there had been evidence of complete arrest of gastric digestion. In one case he had found considerable undigested food in the matter ejected from the stomach nearly nine hours after dinner. There was also evidence that not only the secretory but the motor activity of the stomach was diminished or temporarily arrested. It was probable that the secretory activity of the small intestine suffered in a similar manner.

The first step in the treatment of migraine seizure should be the washing out of the stomach with water at a temperature of not less than 105° F. The chief effect of this treatment is to relieve the pain, and it occasionally aborts the attack. The best results are obtained when the stomach washing is done just at the beginning of the attack of headache. The effect of lavage is better where the stomach contains food, but it should be employed in any case. Where lavage was inconvenient, the patient should drink hot water. While the results were marked and rapid the *rationale* of the method was not so apparent. It was possible that in migraine the tolerance of the nervous system is suddenly exhausted, and hence the removal of a relatively small quantity of toxic material from the stomach may be sufficient to decidedly influence the attack. After the stomach washing the patient should be given a rapid-acting cathartic, one of the best being a tea or dessert-spoonful of Carlsbad salts. His observations would seem to show that in migraine intestinal putrefaction was not an important factor. It was desirable to avoid severe purgation on account of the undue irritation produced. The cathartic should be aided by a hot soap-and-water enema, and this should be given even though there had been a recent stool. If this active treatment were begun within the first hour of the headache it not only markedly relieved the pain but often cut short the paroxysm. When the headache returned it was more easily treated after these initiative steps. Antipyrine he had found unreliable and apt to cause severe digestive and vaso-motor derangement. Phenacetin in doses of ten grains, repeated if necessary, was usually useful. Antifebrin, in doses of five grains, relieved the pain still better, but sometimes acted unpleasantly by depressing the heart. The best of all seemed to be ammonol. This was claimed to be a mixture of an ammonia salt with antifebrin. Black coffee, without sugar,

he had found often very efficacious, and the same might be said of citrate of caffeine. Where the face was regularly much flushed, ergot sometimes acted well, but usually this preparation was not easily retained. He would not care to use nitroglycerine except in cases in which there was distinct flushing of the face. Local applications could only be regarded as feeble adjuncts to the ordinary treatment. It was a pernicious practice to give one of the antipyretics mentioned and not insist upon temporary rest and quiet.

The speaker said that although it might be safely assumed that there was a toxæmia present in severe attacks of migraine we must admit our utter ignorance of the nature of this toxæmia. It was probable that in health the albumoses were absorbed only to a very slight extent. When introduced into the circulation in animals they were found to be much more toxic than peptones. In sufficiently large doses the albumoses were invariably fatal. It was possible that substances identical with, or closely allied to, these substances formed from the digestive process, might be absorbed into the circulation without further change. It was possible that in migraine that there was absorption of pathological albumoses, more toxic than in health. The rapidity with which the headache might be produced in migraine by the ingestion of proteid food would seem to indicate that the attack was brought on by unorganized ferments rather than by bacteria. He could not believe, however, with Haig, that migraine was due to an excess of uric acid or its salts in the blood. Rachford had claimed that migraine was due to poisoning with hypoxanthine, but so far his observations had not been confirmed by others, and they should therefore be considered only as highly suggestive. The theory of toxæmia, however, did not explain the unilateral character of many of the symptoms of migraine and some other phenomena. We must admit, as in epilepsy, that there was an inherited tendency in the nerve cells which rendered them excessively sensitive to the action of such poisons. To this must be added another important factor—fatigue. He was inclined to believe that the digestive derangements and the toxæmia always preceded the headache, although doubtless at the height of the attack the nervous and digestive conditions reacted upon one another. In this connection it should be mentioned that sexual excitement was often a marked exciting cause of migraine. Milk should constitute the proteid food of at least one meal a day, and red meat should not be allowed more than once a day. Many of these patients cannot tolerate fruits any more than gouty individuals. He had known a number of most intractable cases of migraine surprisingly

improved by a change to outdoor life. Horseback riding and bicycle riding he believed to be the best forms of exercise for these persons.

Dr. M. Allen Starr expressed his hearty approval of the paper, and particularly of the therapeutic considerations embodied in it. During the past two years he had given the Rachford method of treatment a careful trial in several severe cases of migraine. The results had been exceedingly good, even in some very obstinate cases in which all the recognized methods of treatment had proved of little value. He had found a few cases in which muriatic acid given at the time of the paroxysm would arrest it. The "Rachford salt" consists of ten parts of phosphate of sodium, four parts of sulphate of sodium and three parts of salicylate of sodium, of which one drachm was to be taken in the morning. As this dose was insufficient to cause purgation he hardly knew to what to ascribe the benefits derived from this treatment. Rachford had insisted upon the necessity of coating the pill of permanganate of potassium in such a manner as to have the pill remain undissolved until it reached the intestine. By means of a keratin coating he had had capsules prepared containing permanganate of potassium and salol. Regarding the diet, he said that it seemed to him very essential to restrict the quantity of red meat. He had found abdominal packs and abdominal massage quite useful in cases of migraine.

Dr. Mary Putnam Jacobi said that a few months ago she had tried on a very severe case of migraine the washing out of the stomach, and had been able in that way to arrest an attack which was unusually severe. In this case the stomach was entirely empty at the time of the washing. The relief was so marked that since then the patient had insisted upon washing out her stomach.

Dr. Frederick Peterson said he was inclined to accept the toxæmic theory of the origin of migraine. He had used the Rachford treatment in a number of cases and had found it unusually successful. One or two patients had rebelled against the Rachford pills on account of the irritation they produced.

Dr. L. Stieglitz said that he could not accept the view that the toxins giving rise to migraine came from the alimentary canal. The periodical cases of migraine often vomited repeatedly, and yet the attack was unrelieved. The hypodermic injection of a small dose of morphine at the onset of the attack often aborted the attack, and as the morphine acted in the opposite way to the treatment advocated by Dr. Herter and Dr. Rachford, the theory did not seem sufficient to explain the phenomena observed. There seemed to him to be a very close connection between epilepsy and migraine. One patient

in his practice had attacks of migraine every eighteen days. It was difficult to believe that the alimentary canal became disturbed and gave rise to toxæmia at such regular intervals.

Dr. Joseph Collins said that in a recent case, that of a woman of thirty-five or forty years, the complaint was of an acidity or soreness in the entire alimentary canal, and of unpleasant sensations connected with micturition and defecation. The paper just presented was exceedingly instructive, as it described most clearly certain clinical features not often emphasized in the text-books. He felt, with the last speaker, that there was a very close genetic connection between migraine and epilepsy. The speculations on the action of the albumoses did not seem to coincide with the treatment advocated in the paper, and also by Dr. Rachford. He had tried the Rachford treatment quite faithfully for two years, but had been unable to find it any more beneficial than the other methods of treatment. He would also say that he had not found ammonol of any use whatever. He had obtained good results however from a combination of fifteen grains of phenacetin, ten grains of salicylate of sodium and five grains of salicylate of caffeine. He had also found a combination of tincture of gelsemium, tincture of belladonna and acetate of potassium very useful in migraine when the vesical distress already mentioned was present.

Dr. Charles Henry Brown said that he had seen a dose of thirteen grains of ammonol in one case cause extreme and dangerous prostration, and he had not observed any benefit from its use in other cases. In the patient referred to, there had been no unpleasant effects observed from the use of antipyrine, antifebrin and phenacetin. In many cases of migraine the polyuria preceded the headache by twenty-four or forty-eight hours, from which it would appear that there was something else besides the toxæmia in the etiology of migraine.

Dr. William Hirsch said that the chief objection to the toxæmia theory of migraine was the occurrence of unilateral symptoms, and the fact that one side of the body alone would be affected time and again. He recalled a case in which a temporary hemiplegia during migraine had finally become a permanent hemiplegia. This could only be explained by repeated vaso-motor spasms, leading to a permanent change in the cortex. At the present time he had under observation a lady who suffered from migraine at each menstrual epoch. The case had proved very obstinate to all the usual methods of treatment, but had yielded best to treatment during the intervals with large doses of the bromides, just as one would treat a case of epilepsy.

Dr. Herter, in closing the discussion, said that notwith-

standing the criticisms that had been made of his treatment he had the greatest confidence in it, for, although ordinarily not very sanguine in therapeutics, he had seen the most gratifying results from its employment. Of course, the patient must be taught to wash out the stomach and attend to the other measures promptly without waiting for the arrival of the physician. As a rule, patients did not vomit until the paroxysm had been well established and absorption was probably going on through a large extent of the intestine, and hence it was not surprising that the eliminative treatment advocated by him did not accomplish much when employed so late. Many of his medical friends had agreed with him that ammonol was a most useful drug for relieving pain and he had not heard of any unpleasant effects from its use. He believed that toxæmias gave rise to hemiplegia much more commonly than was usually supposed; hence this would explain Dr. Hirsch's case. It was the rule for polyuria not to appear until the headache had passed, although occasionally it would persist for a number of weeks.

Periscope.

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CONTRIBUTIONS TO THE STUDY OF THE ALTERATIONS IN THE SPINAL CORD DUE TO PERNICIOUS ANAEMIA.

K. Petren. Nordiskt Medicinskt Arkiv, No. 7, 1896.

In nine fatal cases of pernicious anaemia studied by the author there were found complicating lesions in the spinal cord in two, in four the vessels in the white substance were degenerated, in five petechial hemorrhages were found in the cord analogous to the hemorrhages found in the retina, and in two cases there were chronic degenerative processes in the posterior columns of the cord.

The phenomena presented by these last two cases were as follows:

(1) A woman, thirty-six years of age, died after six month's sickness. The disease began with tactile anaesthesia and was followed later by a loss of all the reflexes. Finally the patient was unable to retain the urine. In the examination the posterior columns throughout the spinal cord showed more or less extensive degeneration. This was most pronounced in the columns of Goll and diminished in intensity from above downward. In the dorsal and in a portion of the cervical cord a state of acute degeneration was found in nearly all of the fibres of the middle portions of the peripheral zone to Lissauer's column. In the dorsal region there was a similar degeneration involving the lateral columns. This had the appearance of a recent and rapid development, and was very diffuse in character.

(2) The other case was that of a man forty-two years old. He was syphilitic and had been hemiplegic for three years and finally died of pernicious anaemia and a disseminated spastic paraplegia. There was found in the cervical and dorsal regions a complete degeneration of the columns of Goll and Burdach, the latter less marked. Degeneration was also found in the crossed pyramidal tracts of the right side. It did not extend below the lower half of the lumbar region, and according to the author's opinion was not caused by syphilitic infection.

JELLIFFE.

CHRONIC ARTERIAL CHANGES IN THE INSANE.—F. del Greco. Manicomio moderno. No. I, 1896.

In one hundred cases of insanity, thirty-two of which had progressive paralysis, and the remainder various morbid psychoses, the

author found that thirty-eight presented arterial lesions (atheroma, arteritis deformans, etc.) united with diffuse changes in the various organs which were more marked in the cases of progressive paralysis.

These vascular changes were regarded as the indications of profound individual organic decadence, and the conditions found mutually influenced one another.

JELLIFFE.

ARTERIO-SCLEROSIS AMONG THE INSANE.

By Dr. E. D. Bondurant. *International Medical Magazine*, July, 1896.

The author gives one of the most valuable contributions to this subject yet produced. He first describes the pathological appearances found, pointing to sclerotic changes in the inner coats of the arteries. The loss of resiliency, the furrowing, the hyaline degeneration, the atheromatous plaques or ulcers and the calcareous plates and fatty changes are all described as various forms of chronic arterial change. He comments on the known prevalence of these among the insane and the more certain knowledge obtainable by post mortem examinations in these cases.

In two hundred consecutive autopsies, he finds in thirty-five per cent. little or no macroscopical evidence of arterial disease, sixty-five per cent. showing distinct arterio-sclerosis; one-half of these show patches in the aorta but not in smaller vessels; the other half show a higher degree of change in the aorta and also changes in smaller blood vessels.

He finds the proportion of this disease slightly greater among the colored people, slightly greater, also among females. As to age, the average age of people showing no lesion was thirty-two. The average age of those with slightly diseased arteries, forty-one; with very marked changes, fifty-five; the youngest patient with well-marked disease was twenty-one; "the youngest showing a high grade of the disease with extensive distribution was forty-three."

Of the causes of death in these cases, among the non-atheromatous, acute diseases, tuberculosis, etc., were prominent—chronic renal diseases being rare. Among the grossly atheromatous, chronic renal disease was "conspicuously frequent," and intra-cranial hemorrhages were frequent.

The valves in the heart were frequently affected, but the heart in these cases was not necessarily enlarged. The kidneys were affected in nearly every case of marked atheroma. In those having little or no arterial disease, kidney disease was less frequent and not pertaining so much to the blood vessels.

In the brain, sclerosis and atrophy were common accompaniments, yet not invariable ones. Miliary aneurisms, so often mentioned, he failed to find. Granular thickening of the ependyma of the ventricle not uncommon. Pia arachnoid usually opalescent, tough, dense, oedematous. Subdural or intrapial hemorrhage is frequent and almost exclusively with arterial disease, yet rarely with gross atheroma.

The clinical symptoms are briefly reviewed. In fifty-five atheromatous cases with autopsies, heart lesions have been noted in eighteen. Arcus senilis was surely accompanied by degeneration. Vertigo and other symptoms are detailed. Poisons in the blood are accorded a prominent causative place; also alcohol, opium and drugs; or infectious diseases, like syphilis, rheumatism and gout; lastly auto-infection and uraemia.

The author gives much consideration to common nephritic disorders of the insane, and makes auto-infection a reason for arterio-sclerosis. He also holds that such poisons, with resultant sclerosis, will invade

many and various organs through the circulation, showing most in that organ where meeting the least resistance.

Finally, mental disease is intimately related to these arterial changes. The cortical cell under such unfavorable circumstances must necessarily suffer, and this can be demonstrated easily enough. The characteristic mental expression of atheroma is a dementia, though not without some outbreak of excitement. PHELPS.

THE CONNECTION OF INTESTINAL AUTOTOXIS WITH CERTAIN COMMON FORMS OF INSANITY. The New York Medical Journal, Oct. 31, and Nov. 14, 1896. By Allan McLane Hamilton, M.D.

Dr. Hamilton injected a number of rabbits with the urine from persons suffering from mental disease, but the results were not characteristic or constant, and he was obliged to abandon the idea that the excreta, in certain forms of insanity, present specific toxic qualities.

He is unwilling to ascribe as much to the influence of uric acid in mental disease as is often claimed. It may be that in certain cases the presence of uric acid is indicative of poisoning due to the destructive metabolism of nuclein, and the initial destruction may be the result of primary intestinal disorder.

He obtained no satisfactory results, as far as the production of symptoms of any kind was concerned, from a series of experiments on rabbits and monkeys with hypoxanthine mixed with the food. It is probable that the intestinal toxalbumins have more to do with the production of disturbance of the nervous system than the leucomaines.

When the gastro-intestinal tract is deranged, certain very virulent toxic agents may be introduced into the general circulation and act mainly upon the nervous system. Especially is this true of the rapidly developing confusional insanities, although in the chronic forms of insanity the occurrence of access and convulsions is attended with some defect of metabolism.

His conclusions are:

1. Urines rich in indican contain very little or no preformed sulphuric acid, and are toxic.

2. When the sulphate ratio is materially changed, it is likely to indicate autotoxis in connection with an increase in the amount of combined or ethereal sulphates.

Such indications are generally found with acute insanities, in which rapidly developing symptoms occur.

4. Fugacious and changing illusions and hallucinations, unsystematized delusions, confusion and verbigeration in connection with insomnia, pallor, intestinal indigestion, constipation and rapid exhaustion are due to autotoxis.

5. Paranoiac states, or those in which concepts are the features, chronic stuporous conditions, and certain forms of dementia have little to do with the formation of intestinal products of putrefaction.

6. Various post-febrile, traumatic, alcoholic, or drug insanities are those in which autotoxis is most constant.

7. The variations in the excretion of combined sulphates keep pace with the changes in the progress of an established insanity, access and epileptoid attacks being directly connected with putrefactive processes.

8. The most successful treatment consists in lavage, intestinal douches, gastric and intestinal antiseptics by means of hydrochloric acid, borax, salicylate of sodium, charcoal, guaiacol, or naphthalin in small repeated doses and in the administration of a combination of the red marrow from the small bones, blood and glycerin.

SPILLER.

PSYCHOSES DUE TO GASTRO-INTESTINAL AUTOINTOXICATION. Wiener klinische Wochenschrift, Nov. 10, 1896. By Prof. Wagner v. Jauregg.

The connection of infectious diseases with polyneuritis is very similar to that of infectious diseases with certain psychoses. It is very probable that polyneuritis may be due to autointoxication from gastrointestinal disturbance. It is not unlikely that those psychoses which resemble the febrile or post-febrile neuritis in their commencement and course are due to bacterial toxin. Psychoses which are similar clinically to these febrile and post-febrile psychoses may have a similar etiology.

Almost all the febrile and post-febrile psychoses belong to the group of acute mental disturbance called by Meynert *amentia*. Frequently patients with *amentia* have had gastrointestinal symptoms at the beginning of the mental trouble, and in some cases these have persisted during the psychosis. When recovery takes place these gastrointestinal disturbances disappear.

Acetone has some connection with derangement of the nervous system (*eclampsia*), but it is probable that acetone is only an indication of the formation of much more poisonous substances. Disturbance of digestion seems to be necessary for the formation of acetone and more poisonous toxins. Wagner believes a relation exists between a high degree of acetonuria and mental disease. In some of his cases, in addition to acetone and diacetic acid, the urine contained a considerable amount of indican. Usually albumin and albumose was found. The acetonuria was not due to fever or hunger in Wagner's patients, although under these conditions the acetonuria is probably of gastrointestinal origin.

Wagner states that the gastrointestinal symptoms should be treated in those psychoses which are due to autointoxication before the changes in the nervous system have become organic. Calomel has given him excellent results. He has also employed iodoform.

He reports a case of acute gastrointestinal disturbance and one of a chronic type with mental symptoms treated with calomel. The indican, acetone and diacetic acid disappeared from the urine, and the patient with the acute psychosis was able to leave the clinic after three days. Recovery in the chronic case was slower but also complete.

[Dercum in his "Address in Mental Disorders" (The Medical News, July, 1895), read before the Medical Society of the State of Pennsylvania, called attention to the various toxic insanities due to disordered visceral action, and recommended the employment of hydrotherapy, not only by means of baths and douches, but also by the injection of large quantities of diuretic waters.]

SPILLER.

THE TREATMENT OF TABES. By Professor Erb. Volkmann's Sammlung klinischer Vorträge, No. 150, 1896.

In treating tabes, the etiology must be duly considered, and syphilis in the first place. As contributing factors, catching cold, forced marches, physical efforts, excesses in "*baccho et venere*," smoking, mental exertions and emotions are enumerated. The fundamental rule of successful treatment is to subject the patient to a prolonged anti-syphilitic cure during the first stages, for, statistics prove that the chances of developing tabes is twenty times greater in those who have not undergone specific treatment. Warm and vapor baths should be avoided. Influenza seems also to predispose to the development of locomotor ataxia in syphilitic subjects. In the controversy as to whether patients, who give a history of previous syphilis, should be

treated specifically, the author considers it as indicated, six-sevenths of the cases showing decided improvement. The anti-syphilitic treatment is specially called for if there are concurrent specific lesions, or if the patient has never undergone an adequate cure. This treatment should not be prescribed in inveterate cases, in dyspeptic or cachetic subjects, in those who have been subjected to it repeatedly without any amelioration, and in patients showing intolerance for iodine or mercury. The old inunction-method with mercuric ointment is recommended, four to six grammes daily, and thirty to sixty rubbings; then the treatment is suspended for several months. The action of KI is less favorable. Tonics and a hygienic régime are also valuable, and if hydrotherapy is resorted to, the temperature of the water must never be higher than 30 deg. C. Every effort must be made to improve the patient's nutrition, and all injurious substances and habits, such as alcohol, tobacco, sexual excesses, exposure, etc., should be avoided. Among drugs, argent. nitr. in doses of 0.03 to 0.5 with or without extract. nuc. vom., is the most useful; strychnine, by the mouth or hypodermically (0.002 to 0.008) has been used with benefit in appropriate cases. The following may also be of service: ergot, the bromides, and arsenic. There is as yet no positive proof of benefit being derived from organo-therapy (spermine, the glycono-phosphates). Electro-therapy is a valuable aid in treatment: the galvanic current along and across the spinal cord, galvanizations of the cervical sympathetic, positive pole applied over the cord in case of pain, and the negative pole along the crural and sciatic nerves. The surface of the dorsal electrodes must be 50 to 70 square cms., the current of medium intensity, and the séances not too long. The electric (faradic) brush over the body and extremities is sometimes used. The effects of static electricity are uncertain. Counter irritations, thirty to fifty fine cautery points around the vertebral column, and blistering are at times beneficial. Among orthopaedic measures, massage and gymnastics may be given a trial. Nerve stretching is at present advocated by but few authorities, while suspension gives good results. Electricity, nervalgia, and local anaesthetics are employed for lancinating pains, and painful visceral crises, but morphine should, if possible, be omitted. Fraenkel has developed a method of muscular exercises which proves quite satisfactory. For vesical troubles, electricity; for tabetic arthropathy rest and compressive dressings of iodine are ordered. Psychological treatment should also be borne in mind.

MACALASTER.

THYMUS GLAND IN THE TREATMENT OF EXOPHTHALMIC GOITRE.

Todd (Brit. Med. Jour., July 25, 1896) reports one of the few recorded cases in which the results of the above treatment were excellent. The patient was a delicate girl of twenty-two, and the disease was well developed and typical in every way. She had been treated at different times with iron, belladonna, arsenic, digitalis, strophanthus and other drugs besides various local applications, without more than slight temporary benefit.

"On September 29, 1895, her pulse was 156 and very irregular both in force and frequency. The apex beat was visible in the sixth space, 1½ inch inside the nipple line. The heart sounds were irregular and tumultuous, but no definite murmur could be made out. The pulsation of the cardiac area was very violent. The thyroid was symmetrically enlarged, with evident pulsation and a loud bruit audible over it. Exophthalmus was marked but not excessive. Von Graefe's sign was not well marked. She was much distressed by the palpitation and suffered greatly from insomnia." Thirty grains of dried thymus

in the forms of tabloids were given daily and on the third day the pulse had fallen to 130 and was quite regular. The treatment was continued, the amount of thymus being gradually increased to 100 grains daily, and at the end of three weeks the pulse had fallen to 73 and was regular, the pulsation over the cardiac area and the thyroid being very much less. The size of the thyroid was not diminished, but the exophthalmos was less marked. The patient felt much better, was able to sleep, and took food well. At one time she was without the drug for three days, during which time she did not feel so well.

PATRICK (Chicago).

TREATMENT OF CHLOROSIS BY OVARINE.

MM. Spillmann and G. Etienne (*Gazette Hebdomadaire de Médecine*, Aug. 27, 1896) treated six chlorotic cases with ovarian substance in the form of fresh sheeps' ovaries or ovarian fluid prepared by the Brown-Séquard method. All the patients suffered acute pain in the lower abdomen with headache and vague muscular aches. Two of the patients had some elevation of temperature; three others improved very distinctly, the general condition grew better, the pallor lessened, the number of white corpuscles increased and the strength improved. Menstruation, which had disappeared for three months and a half, returned after the fifteenth day in one case and after about three months in another. The authors believe that ovarine favors the elimination of toxins, and introduces into the organism an antitoxic principle which has a general favorable effect, a conclusion which is scarcely borne out by the long continued treatment which was necessary before the patients showed any marked improvement.

J. K. MITCHELL.

FURTHER OBSERVATIONS ON THE EXCRETION OF URIC ACID IN EPILEPSY AND THE EFFECTS OF DIET AND DRUGS ON THE FITS. By A. Haig. *Brain*, Spring, 1896.

In this article, the author, whose views on the baneful effects of an excess of uric acid in the system are well known, attempts to establish a causal relation between uric acid and epilepsy. His convictions and his hopes are expressed in the following statement: "The fits of epilepsy . . . bear an extremely close relationship to the uric acid headache (migraine), and like this are probably functional disorders due to altered circulation in the brain. . . . The headache is controlled with almost absolute certainty by a diet which frees the blood from excess of uric acid."

S.

Book Reviews.

A SYSTEM OF MEDICINE BY MANY WRITERS, Edited by THOMAS CLIFFORD ALLBUTT, A.M., M.D., etc., Regius Professor of Physic in the University of Cambridge, etc. Vol. I. New York, MacMillan & Co., 1896. (Price, \$5.00.)

The appearance of a new system of medicine by English authors, the first since that of Reynolds, cannot but excite wide-spread attention. The first volume of the system edited by Dr. Allbutt, is, apart from its merits, interesting in many ways to the American reader.

The introduction by the editor is excellent. Attention is called to the fact that we can no longer properly speak of publishing a "system" of medicine. This work represents, rather, under the present conditions, a statement of "our knowledge set forth on the whole as immediate convenience and the exigences of temporary learning may dictate."

Some good remarks upon types in disease are to be found upon page xxix. There are also, on page xxv., some very judicious observations upon nomenclature, but it will well repay any one into whose hands the volume may come to read the entire introduction.

The volume is divided into two main parts, the first division being devoted to "Prolegomena" and the second to "Fevers."

In one respect this work differs materially from some other recently published systems of medicine. It has been the tendency of late to omit general pathological considerations as far as possible, leaving these for special works; questions of this nature have been taken up separately in the consideration of each individual disease. Here, however, there are nearly five hundred pages devoted to the consideration of general subjects.

Some of the articles are excellent. As is, however, stated in the preface, others are of necessity condensed, and the wisdom of including them in the present volume seems to us a debatable point. Thus, for example, the article upon "Medical Statistics," upon "Temperament," upon "Anthropology in Medicine," upon "Life Assurance," while interesting, are so brief as to be in rather marked contrast to the more elaborate considerations of special diseases which follow. The article upon "Nursing" also appears to us out of place in a volume of this sort.

There is much in this section that is very good. The article upon inflammation by Dr. Adami is a particularly clear and ably presented consideration of the present state of our knowledge and of the various theories upon the subject.

This is followed by an interesting discussion of "The Doctrine of Fever," by Dr. Burdon Sanderson.

Dr. Leach contributes an excellent article upon the "Principles of Drug Therapeutics." The concluding paragraph upon the limits of the utility of drugs is worth repeating; "Drugs only act beneficially when they can exercise such influence on the morbid changes in tissues and organs as to restore the parts to a state compatible with systemic life, but in a large proportion of cases such restoration is impos-

sible. Unfortunately, for the reputation of drugs it is considered necessary to give them in all cases, even where it is manifest that the case is beyond the limits of drug treatment. *The prevalent want of belief in drugs is largely due to the fact that they are expected to achieve the impossible.*" (The italics are ours.)

The section upon "Climate in the Treatment of Disease" by Drs. Weber and Foster is extremely good. It contains much very useful information and is replete with sound common sense. How true is the statement: "The physician is indeed a very important part of a health resort and of a climatic cure, although the invalid is often disinclined to see this. Many lives are needlessly lost by trusting to climate alone." And again (page 292), "Those patients do best who bear in mind that climatic change is the smallest factor in a treatment which is to restore them to health, and setting before themselves the recovery of health as their single aim, submit to a regulated manner of life."

Again (page 297), "If we carefully examine the good results obtained at foreign climatic health resorts, we often find that they are not so much due to the climatic advantages of those localities as to the hygienic and dietetic management and the whole manner of living. We see, for instance, that the results obtained at Goerbersdorf, at Falkenstein and at Hohenhonnef are at least as good as those obtained at Davos, at St. Moritz and Colorado; and yet, in the prominent climatic conditions—namely the elevation, the number of sunny hours, the diathermancy of the atmosphere—the three former localities are decidedly inferior to the three latter; but in these the hygienic and dietetic arrangements, and especially the open air treatment and the limits of exercise are under supervision."

The article contains a detailed consideration of the more important health resorts throughout the world, followed by a brief section upon the utilization of climates and their application to different pathological conditions.

The section on "Artificial Aero-therapeutics," by Dr. Williams, will be found useful. That upon "Balneology and Hydro-therapeutics" by Dr. H. and F. Parkes Weber, will be likewise of much value to the practitioner, setting forth as it does the characters of the different continental baths.

This is followed by a summary of "The Technique, Physiology and Therapeutic Indications of Massage," by Dr. J. K. Mitchell.

The article upon the "General Principles of Dietetics in Disease" deals with matters which, it appears to us, might almost as well have been considered in the sections upon the individual diseases. By this, certain contradictions in the advice which is given with regard to special diets might at least have been avoided. The rules laid down with regard to the diet in typhoid fever are, we believe, rather more rigid than is deemed necessary by most observers of the present day. We must further disagree with the statement that "Champagne can seldom be safely employed in enteric fever." On page 393 the author deprecates, wisely we think, the indiscriminate administration of pre-digested, especially peptonized foods.

Dr. Eustace Smith treats briefly of the "Diet and Therapeutics of Children." We must be allowed to doubt the wisdom of so general a statement as the following (page 421): "After any of the infectious fevers quinine is always indicated. A child of twelve months will take a grain three times a day; and one-half grain may be added for every year of the child's life until a dose of three grains is reached. This can be given three, four or six times a day as may seem desirable. The usual doses ordered for children are too small; for young patients are not at all susceptible to the alkaloid and rarely suffer from cinchonism."

This is followed by an article upon "Nursing" which, we believe,

might better have been omitted. A curious error occurs on page 440, where, in the directions for giving a cold bath, it is suggested that the patient be lowered in a sheet into tepid water (75 deg. F.) (!)

One remark, however, upon page 448, we can heartily endorse.

"One rule would save many difficulties, viz., that both doctor and nurse, *invariably* communicate in writing, never giving or receiving verbal messages from patients or their friends." It would be well had this rule been stretched and applied to verbal orders given by the doctor to the nurse, a source of more danger to the patient and of more misunderstanding between the doctor and the nurse than any point connected with our present system of nursing.

Some judicious considerations upon the "Hygiene of Youth," by Dr. Dukes, follows; while this is succeeded by an article upon "Life Assurance" which is interesting, but, it seems to us, out of place in the present volume.

The second division of the volume takes up the fevers and opens with a consideration of "Insolation or Sun-stroke," by Sir Joseph Fayrer. We cannot agree with the author in his recommendation of the use of large doses of quinine, and we feel yet more strongly that experience has shown the administration of antipyrine, phenacetine or antifebrine to be most unwise. Certainly, no treatment has proven as satisfactory and as free from possible harm as the cold bath judiciously applied.

In connection with the good results which the author has seen in India following quinine, one cannot but remember how frequently pernicious malarial fever may simulate sun-stroke and the converse. Indeed, there are reasons which might lead us to believe that a combination of the two conditions is not very infrequent, an existing malarial infection predisposing possibly to insolation.

The second part of this division treats of "The Infections." In certain respects the division of the infective diseases appears to us not wholly satisfactory. Thus the use of the word "bacteriology" to include the study of all morbidiferous micro-organisms is open to exception. Might it not have been better to employ the term "parasitology" or "microbiology"?

Again, the use of the term "pyococci" in the first section appears to us inexact. Might it not, perhaps, have been wiser to use the safe term "pyogenic micro-organisms"? Moreover, to include under a special heading, "Epidemic Pneumonia," among diseases of "more or less established bacteriology" and to leave the ordinary acute pneumonia for another section—outside of the class of infections?—appears scarcely justifiable.

The first article in this section, that upon "The General Pathology of Infection," by Dr. Kanthack, is an excellent consideration of the subject; it will well repay careful reading.

In Mr. Cheyne's section upon "Septicaemia" no reference is made to the value of examination of the blood in distinguishing the condition from malarial or enteric fevers.

Dr. Dreschfield follows with an article upon "Infective endocarditis," while Dr. Playfair treats of "Puerperal Septic Disease." Dr. Playfair adheres to the old belief that puerperal septicaemia may originate from exposure to the infective agent of scarlatina, believing, apparently that the "poison" of scarlatina may, if introduced through the genital tract, give rise to "an illness indistinguishable from septicaemia." Knowing, as we do, the frequency of grave streptococcus infections (angina) in association with scarlet fever, it might appear more reasonable to account for these instances of puerperal fever in a simpler manner.

The treatment of septicaemia, whatever be its cause, with doses of antipyrine as large as 20 grains, as is advised on page 650, is, we be-

lieve, not only injudicious but harmful. And it is surprising to read, after such advice as that given above, the following statement: "The external application of cold by sponging or by the use of wet towels soaked in ice-water may be tried in very severe cases; but this treatment is troublesome, and as it is palliative and not curative, it is not often called for."

In the following article by Dr. Melson upon "Furuncle," one finds that method which is ordinarily considered to be the best treatment for this condition disposed of in the following words: "Incisions are extremely painful, and few people will submit to this treatment."

Dr. Whitelegge occupies six pages with a discussion of "Epidemic pneumonia"; this is devoted largely to a consideration of the cases occurring in the epidemic at Middlesbrough in 1888 and in the Scotter epidemic in 1890. Though the disease is included among infective fevers of "more or less established bacteriology," the statement is made "that the infection is probably not always of the same kind"—Friedlaender's encapsulated diplobacillus (erroneously referred to as a diplococcus) the Fraenkel-Weichselbaum diplococcus and the bacillus isolated by Klein in the Middlesbrough and Scotter epidemics having all been found. One cannot but question the wisdom of separating these cases from those of ordinary acute pneumonia, while it is difficult to understand the motives which have led the authors to exclude pneumonia from the infectious diseases.

Dr. Ormerod contributes a good article upon "Epidemic Cerebro-spinal Meningitis."

The section upon "Diphtheria," by Drs. Gee, Thorne, Kanthack and Herringham, is very good, while Sir George Humphrey and Dr. Woodhead contribute an excellent article upon "Tetanus."

Dr. Dreschfeld contributes the division upon Enteric Fevers. The section on "Bacteriology" is particularly full, while the clinical descriptions are good. Mention is made of the changes which are to be observed in the blood in typhoid fever, but attention is not drawn in the section upon "Diagnosis" to the really valuable help which, as Uskov demonstrated, may at times be obtained from examination of the blood.

The lack of appreciation of the value of clinical blood examinations in the differential diagnosis of many of the infectious fevers—the presence or absence of leucocytosis, etc.—is not restricted to this article; it is notable throughout the volume.

The author appears to approve of the administration of several large doses of calomel if the case come under observation before the ninth day, a method of treatment which, we believe, is being largely abandoned by those observers who first advised it.

The cold bath treatment is discussed at length and satisfactorily, though we would venture to doubt the assertion that "relapses appear, however, to occur more frequently with the cold bath treatment."

We believe that most physicians in this country who have had extended experience with the use of antipyretic drugs will doubt the wisdom of doses of quinine to the extent of fifteen or thirty grains, of antifebrine 4-8 grains, phenacetine 10-15 grains, or of antipyrine 10-30 grains. Most evidence to-day is certainly against the advisability of the systematic use of antipyretic drugs in typhoid fever.

Considerable space is given to the antiseptic treatment: the author believes that good effects may be observed from the administration of small doses of bichloride of mercury.

The remark upon page 857 that "The preventive against bed-sores is to warn the head nurse that she will be superseded if they occur" is not without wisdom. The article closes with several tables showing the annual mortality and death rate from enteric fever for a

considerable number of years in England and in the Monsall Fever Hospital.

A long article follows upon "Asiatic Cholera" by Dr. Macleod, Mr. Hart, Drs. Smith and Kanthack and Mr. Stevens.

Dr. Payne treats of "Plague," while the subject of "Relapsing Fever," dealt with by Drs. Rabagliati and Westbrook concludes the volumes.

The book, as a whole, is certainly a valuable addition to modern medical literature; it contains much that is extremely good, and one is led to look forward with anticipation to the appearance of the succeeding volumes.

In form and typography the book is admirable, fully up to the standard maintained by the publishers. The shape of the book appears to us to have been happily chosen; it is a great improvement upon the more bulky volumes which have characterized many of the recent systems of medicine.

W. S. T.

INFLUENCE OF ACUTE ALCOHOL POISONING ON NERVE CELLS.

Colin C. Stewart, *The Journal of Experimental Medicine*, Nov., 1896.

In an interesting paper Stewart gives us the results of his experiments on three cats, two of which were subjected to the acute effects of alcohol and one being used as a control animal. He found that there was a progressive diminution in the number of chromophile granules of the cells of the cortical era, of the Purkinje cells in the vermis and of the multipolar cells of the gray anterior horns. Nissl's methylene-blue stain was used for the sections studied, and the results demonstrated by means of a beautiful chart and percentage tables. The number of chromophile granules was much diminished in the animal living but fifty minutes, and still further lessened in that living for fifty and one-half hours, but subjected to much larger quantities of alcohol, injected into the abdominal cavity and subcutaneously. So far these experiments are not conclusive, but serve the valuable purpose of showing, at least, a demonstrable effect of the poison (or fatigue?) upon the large nerve cells of the central system. The work is a confirmation and extension of Dehio's studies upon the same object in other animals. Further experiments on the same and on other lines are promised.

STERNE (Indianapolis).

AN AMERICAN TEXT-BOOK OF APPLIED THERAPEUTICS.—Edited by J. C. Wilson, M.D., assisted by A. A. Eshner, M.D. 8 vol., 1299 Pages. W. B. Saunders, Philadelphia, 1896.

The design of this work is to facilitate the application of the results of the labors of the investigation to the uses of the practicing physician. The articles have been written by American contributors with the exception of that on Malaria, by Professor Laveran, and of that on Leprosy, by Dr. Rakes. The list of writers includes the names of many who have acquired merited distinction as practitioners and teachers of clinical medicine and of the specialties. The arrangement of the book has been based upon modern pathologic doctrines, beginning with the intoxications and following with the infectious diseases due to internal animal parasites, diseases of undetermined origin and finally the disorders of the several systems—digestive, respiratory, circulatory, renal, nervous, and cutaneous. A consideration of the disorders of pregnancy has also been included. Two hundred and forty-three pages are devoted to the treatment of nervous and mental diseases. There is a very full introductory article to this subject, by J. T. Eskridge. Among the causes mentioned are syphilis, abuse of alcohol, tuberculosis, drug habit, heredity, non-marriage, and mental

overwork. He believes regarding heredity, that when a man and woman marry, who are the subjects of vitiated hereditary influences, the physician is justified in advising the prevention of conception. He considers that discretion in sexual intercourse is very important for the reason that if the father is intoxicated at the time of conception the fruit of this event will in all probability be imperfectly organized, especially in regard to its nervous system. We are, he says, justified in believing that any decided variation of the parents from health at the time of conception is likely to affect the child. The remedy is obvious. In referring to the subject of diet, he says—the power of resistance of the body against the invasion of disease is immensely increased by maintaining nutrition at a high standard, for the majority of poorly nourished nervous invalids, a diet of meats, fish, bread, green vegetables and fruit is best. Epileptics, however, do better on a vegetable diet. Regarding tea he says that it disagrees with delicate stomachs on account of the tannin contained, and, hence, it should be prepared so as to be freed from this as much as possible. He allows that tea is also a cause of nervousness and sleeplessness. Coffee, owing to the number and quality of extractive ingredients is considered more trying to the digestive organs than tea, but the quantity of extractive matter may be greatly lessened by allowing boiling water to percolate through the coffee, instead of boiling the coffee and water together. The article on Neurasthenia is written by Wharton Sinkler. The indications for treatment are to promote the nutrition of the nervous system through the improvement of the general bodily nutrition. The causes also should be sought for and removed. A change of air is always beneficial and the morning sponge bath and a course of hydrotherapy are regarded as very important measures. When due to lithæmia the first attention should be paid to the diet and secretions. Alcohol is contra-indicated. Drugs are considered of comparatively little value. Strontium bromide is regarded as the least objectionable of the bromides for affording temporary relief. Hypnotics should be avoided as it is difficult to stop their use when they are once begun. Better results may be obtained from massage, or a warm bath, or some light nourishment before retiring. When the patient is incapable of any kind of exertion Weir Mitchell's rest treatment is considered the most satisfactory.

Chas. K. Mills contributes an article on Cerebral Hemorrhage. He says, as we are dealing here with a patient suffering from the effects of a bleeding vessel absolute rest is very essential. The patient should lie on one side to relieve threatening stertor. Derivatives should be applied to the extremities and active cathartics easy of administration as croton oil should be given together with diuretics.

Nitroglycerin skilfully used, he considers of great value because of its influence on the heart and vessels. In those cases where febrile phenomena come on, supposed to be due to inflammation of the tissues in the vicinity of the clot, it is just as well to direct treatment with this idea and employ local blood-letting, cold, cathartics and bromides. The surgical treatment is also referred to. Other contributors to the treatment of nervous and mental diseases are Sanger Brown, F. X. Dercum, J. H. Lloyd, Guy Hinsdale, J. C. Wilson and J. B. Chapin. Throughout the book it is evident that an endeavor has been made to indicate the course of treatment to be pursued at the bedside rather than to name a list of drugs that have been used at various times. While the scientific superiority and the practical desirability of the metric systems of weights and measures is admitted it has not been thought best to discard the older system, so that both sets are given throughout the text. Certain departures from conventional modes of spelling occur throughout the work, but it is believed

that they are in accordance with prevalent tendencies among scientific writers. There are a number of very excellent illustrations, which materially help to elucidate the text. The index is very complete and it is possible to turn to most any known disease and obtain a full and practical description written up to date of all that is of value regarding its treatment. The type, paper and binding are good, and the book presents an attractive appearance. It certainly is a work which must prove of real value to both the physician and student of medicine.

FREEMAN.

ATLAS DER PATHOLOGISCHEN HISTOLOGIE DES NERVENSYSTEMS.
 Edited by Dr. Victor Babes. Part VI. Berlin: August Hirschwald.

In the first portion of this work Professor Homén describes the histological changes which occur in secondary (experimental) degeneration of the spinal cord. The plates are worthy of the highest praise. The text is very clear and presents the subject of secondary degeneration in detail. Homén made his examination on the spinal cord of dogs. His statements are as follows:

The first signs of degeneration are to be found in swelling of the axis cylinders. These become granular in appearance, do not take the usual stains, but are deeply colored by acid fuchsin. The alterations occur first in the posterior columns above the lesion, and as early as the third day after the operation. At first only a few fibres exhibit any change, and the degeneration attacks these fibres in equal degree throughout their entire length.

Four to five days after the operation alterations may be detected in some of the fibres of the antero-lateral columns below the lesion, and after five or six days degeneration may be seen in the direct cerebellar and Gowers' tracts above the transverse section. The number of degenerating fibres increases gradually, and after fifteen to twenty days there are few normal fibres in the affected areas.

The degeneration of the medullary sheath follows that of the axis cylinder, and is quite far advanced in certain fibres ten to twelve days after the operation. About this time reaction of the neuroglia begins. About the twentieth day karyokinesis is very evident in the neuroglia cells. Dr. Homén presents two beautiful pictures representing this process. The various stages in the formation of corpora amylacea from altered nerve fibres may be observed. Later the degenerated fibres disappear and the neuroglia contracts. Homén in two cords has found a descending degeneration similar to that seen in the comma zones of man, and in a number of cases he has found a triangular area below the lesion on either side of the posterior septum, near the periphery of the cord, which contained degenerated fibres.

Professor Babes in the second half of this work describes the different forms of degeneration and inflammation of the white matter of the spinal cord, of the spinal membranes and roots. A large part of microscopic morbid pathology is included in this description. The illustrations are very fine. As the various processes are very briefly referred to, it is difficult to select portions for special mention.

SPILLER.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ON CERTAIN UNUSUAL FORMS OF PARÆS-
THETIC MERALGIA.¹

By WILLIAM OSLER, M.D.

BERNHARDT, Roth and others have described a remarkable condition of paræsthesia of the thigh, (meros) usually unilateral, and in the area of distribution of the external cutaneous femoral nerve. Roth has written a short monograph on the subject and has reported some fourteen cases (Meralgia paræsthetica, Berlin, 1895), and a number of cases have been recorded in the recent literature (see *Revue Neurologique*, 1895). A large proportion of the patients have been men in the middle period of life.

The symptoms, which as a rule have developed slowly, consist in pain of a burning character and uneasy feelings in the antero-lateral aspect of one thigh. In some instances the discomfort has been so great that the patient has been unable to use the leg, and exercise as a rule has increased it. Tingling, numbness and localized anæsthesia have been present in many of the cases. Roth states that in all of his patients the area of pain has been the same: viz., in the distribution of the external cutaneous nerve. Sometimes the feeling of burning may extend over the whole thigh. In five of his fourteen cases both sides were involved. The condition is more distressing than serious.

¹Read at the Philadelphia Neurological Society.

It has persisted for a variable period, even as long as twenty years. Roth thinks it is possible that in these instances there may be some compression of the nerve, either in its passage under the psoas muscle, or where it runs close to the anterior superior spine of the ilium.

I have not seen a typical case of paræsthetic meralgia, but I have met with three remarkable instances of monocrural paræsthesia, which are possibly exaggerated forms of this trouble.

Case I.—On several occasions in 1890-91 I was consulted by Mr. X on account of a remarkable disability of the right leg. He was a vigorous, healthy man of about sixty years of age, whose family and personal history was excellent. He had been a very hard worker and had lived for years a sedentary life. He had a very prolonged attack of insomnia after his fortieth year, which was only cured by prolonged rest abroad. When he first came under my care he looked a robust, rugged man, in perfect health. His sole complaint was of a peculiar condition of the right leg. There was more or less constantly a feeling of cutaneous irritation or burning, sometimes accompanied with tingling and numbness. The sensation was quite superficial, and when severe he did not like to have anything touch the limb. So aggravated had it been that he had devised various plans to prevent the clothing from coming in contact with the skin. The second feature upon which he laid much stress was an inability to use the right leg without arousing in it the most distressing sensations, which compelled him in a very short time to stop walking.

The patient had seen a number of specialists, all of whom had agreed as to the negative condition on examination. He had studied his own case with a great deal of care and wrote out a long history, of which the following is an abstract.

He described the condition as one of chronic nervous fatigue, or liability to fatigue of the right leg without loss of faculty or function. He ascribed the commencement of the trouble to walking more than usual during the autumn of 1887. The following is his own graphic account of the trouble:

“Early History.—The first striking symptom was a feeling after walking that the legs were stuffed with foreign matter. In December this feeling became so marked that almost any walking excited it, and the patient then

saw for the first time that rest was indicated and massage of legs substituted for exercise."

"In the winter of 1888 he improved so that a moderate amount of walking was indulged in. But in the spring a condition of extreme physical exhaustion set in and continued to increase in spite of rest. It was accompanied by singular feelings in the legs which do not admit of description. One I called the burnt feeling from a fancied resemblance to the feeling of the skin a day or two after an accidental burn or scald."

"About May 15th he took a sea voyage. This tonic removed feelings of exhaustion, but left patient with difficulty of locomotion, owing to feeling of contraction of hip joints or tendons. This showed no tendency to diminish, but seemed not to preclude slow and careful walking to the extent of one-half mile."

"Second Stage.—About May 26, 1888, he took an evening stroll; unexpectedly, the street led up-hill. On slight feeling of being overcome he returned in street-car and went to bed.

"Next morning the left leg had quite recovered from fatigue, but right leg felt as if inflamed and swollen; tingled all over and was so tender that patient had to sit all day with leg horizontal. He consulted a local physician, who prescribed mustard plaster on back on retiring.

"The morning after the leg was enough better to pursue journey home without injury. But a week or so later a walk of one hundred yards brought on another exacerbation, on account of which the patient went to bed for a week.

"June 22 to Sept. 22, 1888: Left leg improved; but right one in such bad condition that locomotion was hardly possible.

"Symptoms.—(a) Feeling of 'tie up,' as if the tendons or fibres were tangled or twisted; so that motion frayed them.

"(b) General weariness in right leg.

"(c) Feeling of heat or inflammation on outside of right thigh.

"(d) Sitting or pressure upon thigh produced feeling of dull numbing pain in both legs.

"(e) Tingling, produced by every kind of muscular action of leg.

Third Stage, Oct.-Dec., 1888.—Change of scene and

mountain air produced great amelioration of all the symptoms, especially a, c and d. Left leg got well; condition of right leg very variable. Neurotic feelings in right leg seemed to leave the thigh and settle in lower leg and foot, which felt abnormal sensations on touch.

"Could generally walk a quarter of a mile without apparent injury other than nervous excitation of leg, which commonly passed off quickly.

Fourth Stage, Jan., 1889 to Dec., 1890.—Gradual diminution of all spontaneous neurotic indications; but no permanent improvement in power to walk. Spells of weakness from time to time of which the patient was quite unconscious till he tried to walk, then supernal and exacerbation, marked by extreme tenderness of nerves, on trying to walk. An exacerbation lasted from four days to a week. No amount of care sufficed to ward it off. In the summer of 1889 mountain air seemed to have lost its power of 1888. During the winter of 1889 a weakening stage seemed to have set in.

Fifth Stage, May, 1890, to Autumn.—In April got still worse; the condition of an exacerbation becoming almost permanent."

It was at this stage that I saw him, and the condition was as mentioned above. The physical examination was negative, there were no changes in the motor or sensory functions. The deep reflexes were exaggerated a little on both sides. The electrical reactions were normal. The subsequent history of the case is interesting. During the following summer he went to the mountains and improved very rapidly. For a year or more he was liable to the attacks, but they gradually wore off, and when last I saw the patient, about a year ago, he stated that the trouble had almost completely disappeared.

In the early stage, as described by this patient, the symptoms suggest the affection described by Moebius as *akinesia algera*, in which painful sensations in the muscles follow the slightest movement, in consequence of which the patients may be bed-ridden and helpless, but the unilateral character and the remarkable paræsthesia are features unlike any affection with which I am acquainted in the literature.

Case II.—A. B., aged 32, seen November 25th, 1893, complaining of numbness and queer feelings in the left leg: of nearly a month's duration.

Patient is a well built, healthy-looking man of good habits and with a very good family history. He had syphilis ten years ago, for which he was treated for three months. The secondary symptoms were slight. He had typhoid fever seven years ago. He is an active, energetic business man, high strung and of a nervous temperament. His present trouble dates from exactly a month ago. One morning before getting out of bed he stretched very actively and got a cramp in the left leg. He thought nothing of it, as he very frequently had had cramp in this leg after stretching forcibly. For a year or more he had noticed this particularly. This morning, however, the pain of the cramp seemed to be more extensive and to affect the entire leg. Throughout the day he had a sort of dull pain in the leg and numbness. He was able to walk, though the leg felt heavy and he favored it a little with a limp. The condition grew worse within a week, and has continued. He has never had any sharp, shooting pains, but a dull, heavy numbness extending throughout the entire limb. There has been no return of the cramp. He says that when he grasps the leg it feels quite different from the right, and gives him a stinging sensation. He has been very nervous and much concerned about it, but it has not kept him awake at night. The chief difficulty has been that it has prevented him from walking actively, that he limps a little and at the end of the day he has a very tired feeling in it.

When stripped there was no difference to be noticed between the two legs, no wasting. The station was good, the muscular power was perfect, the reflexes were equal and both knee-jerks were a little more active than normal. The skin on both legs seemed normal, the temperature of both was the same. The soles of the feet and palms of the hands were moist. Tactile sensation was everywhere perfect. He recognizes instantly if the hair on any part of the leg was touched. On grasping the legs, too, he felt no difference on the two sides. The pin prick was everywhere felt, and there was not the slightest retardation, but he did not feel it as pain. On the left leg he did not distinguish promptly between the hot and cold test tube. He did instantly on the right leg, but on repeated trials with the left leg he always said that the warm glass felt a little more soothing, but he did not clearly distinguish between the heat and cold. There were no special areas of

anæsthesia or of paræsthesia. He walked perfectly well about the room, but expressed himself that the leg felt heavy and dull and tired easily, and that he was always conscious of its presence.

In the groins the lymph glands on both sides were a little enlarged, the pulsation in the femoral arteries was equal. There were no nodular masses to be felt in the pelvis.

Everywhere over the surface of the skin the scratch of the fingernail was followed by a very active reddening. The patient was ordered cold douches and massage to the back and to the leg.

I saw the patient again on December 5th. He limped a little; why, he did not know, but said that it was easier for him to walk so; he then had less fatigue in the left leg. He expressed himself as feeling very much improved by the treatment, and said that the sensations in the leg were very much better, and now felt quite natural over the greater part of the foot and on the side of the ankle. To-day everywhere over the left leg he feels the prick of a pin as a painful sensation.

I lost sight of this patient until November of last year, just two years after the attack. He reported that the numbness had disappeared, but that he occasionally had the cramps in the leg at night.

Case III.—Mr. H., aged 37, professor in a southern college, consulted me June 22d, 1896, complaining of trouble in the right leg when he walked. The patient was a very nervous man, who had worked hard, often too hard, and had frequently had attacks of nervous dyspepsia. He has had no serious illnesses except the common affections of childhood.

He states that about four years ago, when very nervous and having frequent attacks of vertigo, he noticed a curious disturbance of sensation in the hollow of the right foot. On walking or standing for any time the spot would become rather numb and feel as though some fluid were rapidly evaporating. Then a sensation of numbness and lifelessness would gradually extend over the foot and leg, almost to the knee. If he sat down and rested the numbness and uneasy sensations lessened. If he persisted in walking the feeling would increase and the leg would become so weakened that he would be compelled to sit down. After resting a few minutes it would pass away.

He noticed it particularly in cold weather. He rarely had any trouble when at rest, though at night sometimes the legs would seem very tired. He describes the sensation very accurately as a burning and a numbness. It is general, and when he has persisted in walking it extends over the leg and thigh, and is as bad in one place as another. Frequently, if he persisted in walking, there was a feeling of burning and contraction in the stomach, sometimes, as he expressed it, a "sick feeling," and a sensation up the back as though the pain was rising. His description of his sensations after walking any distance is almost identical with that given in Case I. In a recent note he states that he can now walk with much more ease, but a distance of half a mile will make the foot very troublesome.

The physical examination was negative. He was a thin, nervous-looking man; the pupils were of medium size, reacted actively. There was marked dermatographia, and the tendon reflexes were everywhere active. The legs were of equal size, mobility in all the muscles good. There was not the slightest disturbance of any of the forms of sensation and the skin felt natural. There was no tenderness along the course of the nerves. There was nothing to be felt on deep palpation in the pelvis.

A STUDY IN APHASIA.

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(Continued from page 97.)

A full understanding of the function of Broca's centre necessitates the knowledge of its relations not only to articulate speech but also to the faculties of reading and writing.

It will be remembered that Wernicke⁹ compared the process of acquiring speech to a reflex process, saying that each auditory impression deposited in the so-called auditory speech centre evoked the corresponding motor concept required for the utterance of the corresponding sound or word. The part which visual, tactile and muscular or kinæsthetic sensation may play in the acquisition was not at all considered by this author. Only Wyllie has called attention to this point.

A careful analysis, indeed, shows that the visual, tactile and kinæsthetic elements have a much more direct influence upon the acquisition of speech than the auditory element.

The importance of the visual impressions is suggested by the fact that those articulatory motions which the child can most easily observe in others, namely, those acquired for the utterance of the lip sounds, as "papa," "mama," etc., are first learned, and that those which escape direct observation, as for instance, the k and the German ch, cause often the greatest difficulty. When we teach a child to repeat a word, we can notice that it looks attentively at the speaker and observes the motions made in uttering the sound.

Let me quote another no less characteristic instance.

⁹Wernicke: Der aphasische Symptomencomplex. Breslau, 1874.

The German *r* differs from the English *r* in sound. While the German *r* is produced by holding the apex of the tongue against the anterior part of the hard palate and letting it vibrate in this position, the enunciation of the English *r* requires an upward and backward rolling of the tongue during the vibration. The German may live among English-speaking people without ever learning the correct enunciation of the *r* and, principally for this reason that, although hearing the difference of the two sounds he does not know and especially not see wherein the difference of the required motion of the tongue lies. I, for my part, only learned to enounce the English *r*, at least approximately, after the backward rolling of the tongue had been demonstrated to me *ad oculos*.

But, of course, tactile and muscular sensations must be added to the visual impressions in order to enable the correct execution of the sound.

Let me further call attention to the fact that when we foreigners are taught to articulate the English *th*, the teacher shows us in what position the tongue has to be held and what motion it has to perform.

The part which the various sensations play in the acquisition of articulate speech may be expressed in the following manner:

"The auditory element serves largely to inform the person speaking whether his utterance is such as he intended it to be, while the visual, tactile and kinæsthetic elements, by modifying motor innervation, help to make such utterance possible and by degrees more and more correct." It is evident that a complicated process of co-ordination and fusion must occur here. We must further assume that Broca's centre participates directly or indirectly in this process and that the products of the fusion, the psycho-motor images of speech, are deposited in this centre.

Each word that we utter aloud is heard by us; but even if we speak only mentally, inwardly, we equally receive a distinct auditory impression of the internal utterance. In other words, each innervation of Broca's centre is conveyed also to the centre for the auditory word images, and every other sensory image which becomes associated with a psycho-motor image of speech, is at the same time associated with the corresponding auditory image. It

cannot be said on the other hand that each innervation of the auditory centre is carried to Broca's centre, as the auditory image of a word or letter need not necessarily arouse the corresponding psycho-motor image. We shall see presently that these facts are of importance for the understanding of the mental process by which we read and learn to read.

The task imposed upon the child who learns reading is to associate the visual image, say, for instance, of the letter "c," both with the psycho-motor concept required to utter the sound "c" and with the auditory image of this letter. It may be seen from the foregoing statements that the association of the visual image with the psycho-motor image of the letter "c" implies that the former is associated with its auditory image, since the latter is invariably coinnervated with each innervation of the psycho-motor image. A direct association between the auditory and the visual image seems, therefore, not absolutely necessary for the mental act of reading, and it may even be doubted whether such direct association becomes established. It is very probable, on the other hand, that a direct association forms between the visual and the psycho-motor image, which latter then coinnervates the auditory image. The understanding of the text read is brought about by an analysis of the series of auditory images evoked in the manner described.

Self-observation convinces us that the innervations take place in the serial order described. When we read, we have the distinct impression that not the "sounds," but the psycho-motor images of the words are first aroused and only secondarily the sounds." In other words, the visual image is directly transmitted into internal language, no matter whether we read aloud or mentally, and the symbols of written or printed language have only a meaning for us in connection with the internal language; they lose their meaning, as soon as the internal becomes impossible.

It is evident from these facts, that although the mental analysis of the text read occurs from the auditory images, the faculty of reading depends in the same degree upon the intactness of Broca's area as it does on the intactness of the centre of the auditory word representations. As soon as the visual image cannot be transmitted into inter-

nal language, both loud and mental reading become impossible. For this reason lesion of Broca's centre must cause, not only motor aphasia, but also alexia, not only the impossibility of loud reading but also that of mental reading; the characters and words are perfectly without meaning for such patients. On the contrary, subcortical motor aphasia leaves the internal language and, consequently, also the faculty of mental reading intact, while loud reading is, of course, also impossible in this form of aphasia.

Dejerine¹⁰ and Mirallié have come to the same conclusions on the ground of ample clinical evidence. They tested the faculty of reading in fifteen cases of cortical motor aphasia. In all these cases disorders of mental reading (*troubles de la lecture mentale*) were found. Eight of these are now cured of their alexia and three of them cured of their aphasia. At the beginning of the disease the letters had no sense for them, they saw only black on white. In the seven other cases there are still distinct disturbances of mental reading and in none of these cases speech has become normal as yet. Three cases of cortical motor aphasia were observed by one of these authors in highly cultured individuals of very high social standing, and in all three patients the alexia was present in a very marked degree.

The manner in which a child learns to read written signs does not differ from that in which it acquires the knowledge of printed characters, but of course both kinds have to be learned if the child is to be able to read and write written characters. The technique of writing could be learned to perfection without knowledge of the letters and words as symbols of language. The motor writing concepts can, therefore, be acquired without any association with the speech mechanism. Such association must occur, however, in any kind of writing except purely mechanical copying.

Part of the process underlying spontaneous writing is probably identical with that underlying writing by dictation, in this sense, that we mentally dictate the words we

¹⁰Dejerine et Mirallié: *Comptes Rendues de la Société de Biologie*, 1895, p. 523.

intend to write. In spontaneous writing the motor-speech centre is started into action by the concept which we wish to express. From the motor-speech centre the impulse passes to the centre of the auditory word representations, evoking the auditory image of the word to be written. The auditory image excites the corresponding graphic concept.

In writing from dictation we first receive auditory impressions, "sound images" are deposited. The further task is, to fix the chain of auditory images received, to remember the words or sentences heard. This fixation is evidently established by help of the internal language. We inwardly repeat what we heard and by a mental analysis establish landmarks, enabling us to reproduce the auditory impressions received in the correct serial order. The further steps are the same as in spontaneous writing. We mentally dictate to ourselves the text remembered by innervation of the motor-speech centre, etc.

It may be asked whether this process must invariably be gone through, or whether it may be shortened in such manner for instance, that the auditory images of the language dictated would evoke directly the corresponding graphic concepts. Such explanation must be excluded *a priori* where whole sentences are concerned. It cannot apply either to the writing of whole words to dictation. The writing of a word implies the ability to spell it, as we must write down the single letters composing the word in correct serial order. In other words, we must also mentally dictate to ourselves each single letter of the word, which again requires the co-operation of the motor-speech centre. For the writing only of single letters to dictation the possibility remains, that the auditory image of the letter evokes directly the corresponding graphic concept, and that consequently in this case the co-operation of the motor-speech centre may eventually be dispensed with.

Quite similar reasonings apply to the faculty of copying. Every kind of copying except that which is purely mechanical, as for instance the copying of perfectly foreign signs (say, Chinese), requires the transmission of the signs into internal language. Consequently cortical motor aphasia implies not only alexia but also agraphia, with this restriction, that the faculty of such purely mechanical copying as is equal to the copying of perfectly foreign characters, is

preserved, and probably¹¹ also the faculty of writing single letters to dictation.

Freud¹² has recently called attention to the important part which functional disturbances play in the causation of aphasic disorders, even in those cases where organic lesions are the primary cause. Bastian's theory on the various degrees of diminished function of a centre has partly served as a basis for his deductions.

Bastian¹³ distinguishes three states of lessened excitability of a centre. The slightest reduction of function expresses itself in such manner that this centre does not respond any more to volitional excitation, but can still be excited by association from one centre to the other, as also by direct sensory impulse. In the second degree of functional disturbance the centre responds only to direct sensory stimuli, while in the most severe or third degree even sensory stimuli fail to excite its functions.

Freud conceives the anatomical arrangement of the cortical speech mechanism as follows: "The speech apparatus presents itself as one continuous cortical district of the left hemisphere, situated between the cortical endings of the auditory and optic nerves and of the motor-speech and arm fibres. Any lesion within this zone of language will display its influence not only upon one single function, articulate speech for instance, but will also debilitate to some degree all the other functions of language, producing one of the states referred to by Bastian.

Freud claims that the various components of his zone of language, such as Broca's area and the centres of the auditory and of the visual word-images, are connected with one another by means of fibres which do not leave the cortex in their course. He therefore concludes that

¹¹This view is confirmed by the investigations of Thomas and Roux (Travail du service du Dr. Déjerine. Bull. Soc. Biol., 1896, p. 210-213). In three cases of cortical motor aphasia these authors found that the patients were absolutely unable to write whole words to dictation; but they could write them, if each letter was dictated singly. This fact makes it appear as not only probable, but almost certain, that the faculty of writing single letters to dictation remains intact in cortical motor aphasia, as long as the motor-graphic memories are unimpaired.

¹²Freud: Zur Auffassung der Aphasien. Wien, 1891.

¹³Bastian: On different kinds of Aphasia. Brit. Med. Journal, Oct. 25th and Nov. 5th, 1887.

Brain as an Organ of Mind. International Scientific Series, 1880.

every aphasia must be due to disturbances within the cortex itself, saying that subcortical lesions may cause dysarthritic disorders but no actual aphasias. This view must be considered as erroneous, since, on the contrary, subcortical disease may produce the purest type of motor aphasia.

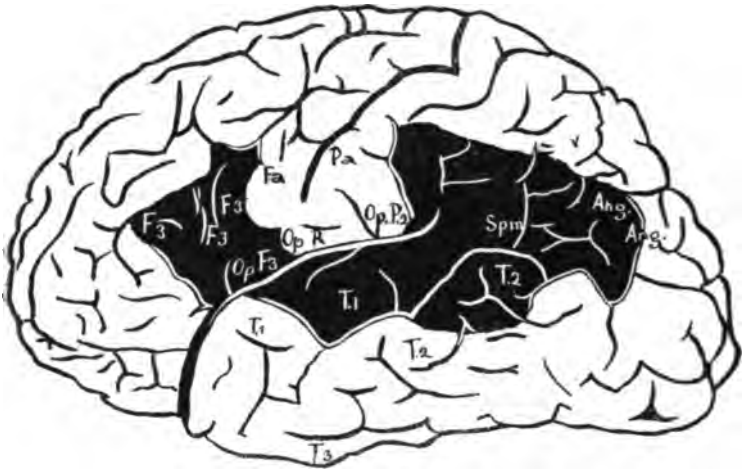


Fig. III.—Drawing illustrating Dejerine's zone of language, reproduced from Dejerine's drawing

■ Zone of language.

Fa. Anterior central convolution.

Pa. Posterior " "

F 3. Third frontal " "

Op. F 3 Opercular portion of the third frontal convolution.

Op. Pa 2. " " inferior parietal " "

Op. R " " central convolutions.

T 1. First temporal convolution.

T 2 Second " "

T 3 Third " "

Sp. m Supermarginal gyrus.

Ang. Angular gyrus.

Mirallié and Déjerine¹⁴ accept Freud's view of a continuous zone of language, within the dominion of which fall Broca's centre, the centre of the auditory word memories and that of the visual word memories. But these authors ascribe a smaller extent to this zone than Freud does.

¹⁴Mirallié et Dejerine: *L'Aphasie sensorielle*. Paris, 1896.

It is beyond doubt that the three centres mentioned are in such intimate functional relations with each other that lesion of one of them invariably tells upon the others. Thus alexia is caused not only by lesion of the centre of the visual memories but also by that of the psycho-motor images of speech; agraphia is caused not only by disease of the centre of the auditory word memories, but also by alteration of the motor speech centre; the internal language becomes impossible not only by lesion of Broca's centre, but also by lesion of the centre of the auditory word memories, etc.

Yet this physiological relationship between the said three centres does not necessitate an anatomical contiguity or continuity of the three. Elder's observation speaks indeed against such continuity and against the view of a contiguous and continuous zone of language in general, since in his case in spite of an extended defect in the central part of Freud's and Dejerine's zone of language, no aphasic, but only dysarthritic disturbances were present.

In returning to our case let us see what application we can make of the deductions arrived at. It will be remembered that at the time of the first examination the patient presented only slight disturbances of speech, but very marked disorders of the faculties of reading and writing. According to the patient's report, however, she must have had a perfect loss of the faculty of motor speech and she began to regain its use as late as two and a half months after the apoplectic attack causing the aphasia and the symptoms accompanying it. It has been shown to what an extent the functions of reading and writing depend on the intactness of the so-called motor-speech centre. We can, therefore, easily understand the coincidence of the motor aphasia with alexia and agraphia in our case, if we assume that the lesion underlying these disturbances involved Broca's centre. The absolute intactness of vision and of the visual memories excludes the possibility of a lesion in the occipital lobe. The view that a lesion of Broca's centre gave origin both to the aphasia and to the alexia finds further support in the fact that many cases of motor aphasia with alexia have been reported in which the aphasia had been almost entirely recovered from, while the alexia remained. The question is, Why does the alexia in these

cases, although caused by the same lesion as the aphasia, remain longer than the latter. The answer might be suggested by the fact that normally the faculty of reading is built up upon the faculty of speech, as the child first learns speaking and then reading and writing. We might conclude in general that the memories of the muscular innervations required for the execution of articulate sounds are called up easier from the sound images than from the visual images of letters and words, and that the sound memories consequently form a firmer basis for the re-acquisition of the motor-speech memories. It must further be kept in mind that by hearing others speak the patient was constantly receiving auditory impressions without any voluntary effort on her part, while to re-learn reading would require special efforts, which in our case were certainly not made, as the patient never made any attempts at reading and writing after she had discovered her inability in that direction.

Although this explanation seems rational, it is contradicted by Déjerine and Mirallié's¹⁵ experience, according to which the patients affected with cortical motor aphasia usually recover sooner from the alexia than from the aphasia, being still distinctly aphasic at a time when the faculty of reading is fully restored. I must, therefore, leave the question unanswered why in our and many other cases the aphasia was sooner recovered from than the alexia.

In discussing the extent and nature of the lesion of Broca's centre two possibilities must be chiefly considered.

First.—The lesion might have been very extensive, destroying nearly the whole centre. This possibility may be rather safely excluded since it is highly improbable that in such cases an adult should so fully recover articulate speech within eight months after its loss.

Second.—The lesion of Broca's centre, which gave rise to the motor aphasia and the alexia and agraphia dependent upon it, might have been small or non-destructive. According to Freud, this would not cause a partial motor aphasia manifesting itself by the loss of a limited number of words, "but by a reduction in toto" of the entire motor speech centre. This functional reduction might show itself under one of the forms described by Bastian. The

¹⁵L. c.

motor-speech centre might, for instance, still be called up by association from other centres, but not by voluntary impulse; or they might still be called up from one centre, for instance, from the centre of the auditory images, but not from another, for instance, not from the centre of the visual word memories. Such a state could even have been produced by collateral changes due to a focus, not of the motor-speech centre itself, but of its vicinity. In our case the association of the motor-speech memories with the auditory memories of speech was already restored when the patient was first seen; but the association of the motor-speech centre with that of the visual word memories and its indirect association with the graphic motor memories was still defective; the patient, therefore, still presented distinct disturbances of the faculties of reading and writing. But these disturbances made indeed the impression, not of destroyed, but of only weakened function. This seemed evident from the following facts:

Neither the letters nor the words seemed absolutely foreign to the patient. She frequently did not recognize a letter or a word at first, but after long deliberation found it out correctly. She would be unable to read a letter in one word and would recognize it a few minutes afterwards in another one. She would further frequently manifest her inability to read a letter, not by saying that she did not know it, but by the remark, "I forget." Three letters, viz.: q, v and x, which could not be recognized spontaneously, were found out correctly by reciting the alphabet until the letter in question was reached. The fact finally that she made much more rapid progress in reading than could be expected if this faculty had to be acquired entirely anew, corroborates the view that the cerebral mechanism presiding over the said faculty was not destroyed but only weakened in its function, presenting one of the states referred to by Bastian.

The findings of the case reported offer opportunity for referring once more to the psychology of the act of reading. Wernicke¹⁶ and Grashey¹⁷ declare that reading is done spellingwise. The observations in our case would tend to prove that this statement is erroneous or too far

¹⁶L. c.

¹⁷Grashey: Ueber Aphasie und ihre Beziehungen zur Wahrnehmung. Arch. f. Psych., Bd. XVI., p. 654.

reaching, at least with regard to the English language. That our patient read most of the words as a whole, not by spelling, was shown by the following facts.¹⁸

1. Entire words were read more promptly than letters composing them.

2. Words which were read correctly were spelled wrongly.

3. The patient often spelled a posteriori from the sound of the word ("one" read correctly but spelled w-o-n, "unknown" read correctly but spelled u-n-w-n-o-w-n). In arriving at their conclusion that reading always occurs by spelling, Wernicke and Grashey evidently did not consider the peculiarities of the various languages, especially of the English. The varying enunciation of the same combination of consonants, or of vowels or of both, make it impossible to read English only spellingwise. It is enough to call to mind the threefold enunciation of the "ough," of the double enunciation of "ow," of the threefold manner of writing the sound "n" (n, kn, wn, etc.). It may further be mentioned that a new method of teaching is now in use in many schools, by which the children are taught to read words before learning the single letters. This method seems indeed more rational, as the

¹⁸The examination of the patient was made before I had read Thomas and Roux's interesting observations on the latent disturbances (troubles latents) of mental reading (*Travail du service du Dr. Dejerine à la Salpêtrière. Bull. Soc. Biol.*, 1896, p. 210-213). The tests proposed by these authors were therefore not applied.

T. and R. conclude as follows:

"The alexia of the patients affected with cortical motor aphasia has some special characteristics. It is rare that the patient with cortical motor aphasia is completely alexic. He most frequently recognizes his Christian name, the name of his children, the name of usual objects. This number of "usual names" varies in proportion to the degree of the patient. The usual words are read, the unusual words are not understood by them, even if they are able to spell them.

"If the letters composing a usual word were written one above each other and at some distance of each other, but in the correct serial order, the patients were not able to read it, even when they could spell each single letter. They could read the word immediately, if it was presented to them in the usual aspect, that is with horizontal arrangement of the letters. But they also failed to understand a word, if each single letter of it was called out to them, one after the other. The authors refer these troubles directly to a change or complete loss of the faculty of mental spelling. 'This change or loss is, on the other hand, nothing but the consequence of the alterations of the associations between the auditory and the motor images.' That our case would have revealed similar disturbances, if the corresponding tests had been applied, seems highly probable."

visual word memories become thus directly associated with the formerly acquired sound memories and psychomotor images of the words. But even if reading was learned purely spellingwise one would learn to read as a whole those words which occur frequently. Many persons will read a language correctly and with great fluency and yet make numerous orthographical mistakes in writing, for the reason that certain combinations of letters are read as a whole and only the sound of them is remembered. "Enough" may be written "enuff," "though" may be written "thow." He who has learned a new language by grammar is less apt to make orthographical mistakes in writing than he who learned it directly from hearing; yet the latter may eventually read it much better than the former. I had opportunity to make some self-observations when I was learning Russian. The letters of this language were entirely foreign to me and I began to read before I was entirely familiar with them. Even now, if the single letters of the Russian alphabet were shown me one by one, out of alphabetical order, I should probably not recognize some of them at all, but if whole words were shown me I could read most of them without mistake, except in accentuation, because I should recognize some of the letters and half guess the rest from the sense. Although to a certain degree I had to learn the reading spellingwise, the memories of whole words read have become more firmly established than those of the letters composing them. I can easily read the words "Bechterew, Kowalewski, Neurological, Neuropathologist, central nervous system, university, and more words which, taken together, contain all the letters of the alphabet. I can read them easily because they occur frequently in the text I read. I can single them out on a page, yet when a new word occurs I have great difficulty in deciphering it. I may add, too, that, although the Russian types were perfectly foreign to me, my knowledge of the Polish language made it much easier for me to learn to read Russian than it would have been to an Englishman, Frenchman or German, because the Russian language resembles the Polish much in sound, although in type it differs materially from it. In the Polish language Latin characters are used.

It remains to explain one peculiarity of the case, name-

ly, that the patient invariably used printed characters instead of script in writing. I believe this peculiarity is due to the loss of the motor-graphic memories, id est of the memories of the motor concepts presiding over the technique of writing written characters. Pitres,¹⁹ Charcot²⁰ and others claim the existence of a special graphic centre, homologous to the motor-speech centre. Some authors locate this centre in the second frontal convolution. Sachs²¹ and others consider it as a part of the arm centre.

Pitres reports a case of motor agraphia which is of especial interest, as it presented this disturbance in its purest type. It concerned a patient who was struck with hemiplegia, loss of consciousness, etc., with subsequent aphasic symptoms. The attack was probably of syphilitic origin. The general symptoms yielded promptly to mercury treatment, the local manifestations subsided gradually. Half a year after the attack the patient could walk without aid. One year and a half after the onset of the disease he came under Pitres' observation, who found the following condition:

"No disturbances whatever of speech and of reading. The right hand shows only very slight motor disturbances, yet the patient is unable to write anything spontaneously or from dictation with this hand. With the left hand he writes quite well from copy, to dictation and spontaneously. The word "souvenir" is dictated and the patient asked to write it with the right hand; he is unable to do so, but he writes the word with the left hand and is then able to copy with the right hand what the left has written. This alone proves that the impossibility of writing is not due to any mechanical difficulty. He can also copy with the right hand what somebody else writes in script. If, however, he has to copy with this hand from printed text he can only reproduce it in print-types, not in script, while in copying with the left hand he directly transmits the printed characters into written signs.

Pitres justly explains the case of assuming loss of the memories of the combinations of muscular efforts required

¹⁹A. Pitres: *Considérations sur l'agraphie à propos d'une observation nouvelle d'agraphie motrice pure*. *Revue de Méd.*, 1884.

²⁰Charcot: Quoted from Pitres, l. c.

²¹Sachs, l. c.

for the tracing of written characters. He further concludes that as a distinction is made between glossoplegia and logoplegia there should also be one between brachoplegia and graphoplegia, and motor agraphia should be considered as a homologon of motor aphasia.

Our case differed from that of Pitres in as much as our patient showed the same graphic disturbances, although in less degree, in the left hand, which Pitres' patient presented in the right hand. In our case there was such an extreme degree of paralysis of the right hand and finger-muscles with extreme contracture of these parts, that writing with the right hand was out of the question. If by some mechanical contrivance the patient would have been enabled to write with the right upper extremity it would only have been a writing with the whole arm: Such test was not made and would not have been of much clinical importance.

With the left hand the patient wrote only in printed characters both from dictation and in copying from printed text. Although she was not absolutely unable to reproduce script, she had so much more difficulty in using written signs that she never attempted to do so except on special request. In most languages the signs commonly used in print differ from those used in writing. Most people read principally from printed text while they write in written characters, which for English-speaking people are like the Latin. Written symbols are much less frequently read from than printed characters, for which reason the visual memories of printed signs are on the average much better established than those of script. On the other hand, by using only script in writing we develop by practice a special technique, a great skill and rapidity in the execution of these characters. The acquirement of such skill is due chiefly to a fine organization of that cerebral apparatus which presides over the co-ordination of the graphic motions. This co-ordination is guided partly by visual impressions, as we can control our graphic movements with the eye, but still much more by muscular sensations. That the latter play a very important part is evident from the fact that we can write quite well with the eyes closed, that we make very few mistakes in so doing, and that the technical execution of the letters and words thus written is nearly as good as when we write with the

eyes open. The muscular sensations accompanying our graphic movements leave memories which with practice become more and more strengthened and enable a rapid selective innervation of certain groups of cells, the combined excitation of which will produce one definite movement; they also enable a serial innervation of the whole series of muscular efforts required to execute a particular letter. If we are warranted in speaking of a motor-speech centre, we must also be justified in speaking, if not of a centre, yet at least of a physiological apparatus in which the said muscular graphic memories are stored. Loss of these memories need not imply the absolute impossibility of writing in written types, as the visual memories may supply the loss sustained. Usually, however, the visual memories of written types are very dim, 1st, because written types are, as a rule, much less frequently read from; 2d, because they are usually much more varying in shape, since one person has an absolutely different character of writing than the other and since even the same individual may show a great variety in the execution of one and the same letter. Consequently, individuals who have lost the muscular graphic memories will show great difficulty, if not inability, of executing written characters spontaneously or from dictation, while the faculty of purely mechanical copying such characters will be preserved. These disturbances will, of course, be much less marked in a person who does much writing and much reading of written text. Here the assistance of the visual memories will greatly mask the loss of the muscular memories.

Ordinarily, we learn to write with the right hand, and thus develop a graphic mechanism in the left hemisphere. Our first trials to write with the left hand succeed badly, as we have only visual memories to guide us. But if the hand becomes disabled, say from a fracture, for instance, we may learn to write with the left hand, as well as we did with the right, by developing a graphic apparatus in the right hemisphere. This, probably, explains the condition in Pitres' case. The patient, after having fully recovered the faculty of speech, began to practice the left hand for writing when his right one was still much paralyzed. Our patient had never tried to write with the left hand. If she had, she probably would have attained the same faculty as Pitres' pa-

tient, and the latter would probably also reëstablish a motor-graphic apparatus in the left hemisphere, as he possessed the mechanical faculty of executing the characters with the right hand, if he did not find it more convenient to continue using his left hand, with which he had evidently attained considerable practice.²²

In speaking of a graphic-motor apparatus it is not meant to convey the impression as if such formed an anatomically circumscribed centre. There can only be question of a physiological, not of an anatomical, unity. In other words, it is not supposed that a lesion may just destroy the motor graphic memories and leave intact all the motor memories presiding over other technical faculties of the hand and finger muscles. On the contrary, it is supposed that such other memories are stored at least partly within the same area, presumably the arm centre and its vicinity. But there must be anatomically to a certain degree a separation between a more elementary and a higher organized motor apparatus, the latter being perhaps represented in the superficial, the former in the deeper strata of the cortex, presumably in the large pyramidal cells.

Some remark is still in order regarding the effects of lesions of Broca's centre upon the high mental faculties. It is usually supposed that loss of the motor speech memories need not cause any disturbances of intelligence. This view seemingly finds support in the fact that deaf mutes, who do not develop a motor-speech centre can yet exhibit high degrees of intelligence. This, however, only proves that the mental processes of these individuals become otherwise arranged, otherwise organized. Their higher conceptions are chiefly built up from visual impressions, and they express their thoughts chiefly by muscular and visual memories. As the motions of the fingers form a very essential part of the apparatus by means of which

²²Dejerine interprets Pitres' case otherwise. He ascribes the *agraphia* to an interruption of an association between the arm centre and the angular gyrus of the left hemisphere, which implies that according to his view the graphic images are innervated directly from the visual images. It is much more probable, however, that they are evoked from the auditory images in the manner described on page III. The *hemianopsia* which Pitres' case presented could not explain the *agraphia*. As this *hemianopsia* was not accompanied by *alexia*, it is difficult to conceive that it should have been in causal connection with the *agraphia*.

deaf mutes express their thoughts, a centre probably forms which assumes such a dignity over the arm and, especially, the hand centre as in normally organized brains the speech centre does over the face, tongue, larynx and pharynx centres. In a person with a normally organized brain, however, the process of thinking occurs predominantly by way of the motor and sensory speech centres, we think chiefly speakingly. The synthetic mental faculties especially are greatly dependent upon the intactness of the speech apparatus. It has been shown that in reading the visual word memories innervate directly the motor-speech memories, which latter evoke the sound memories of the words. "We speak inwardly what we read," and can only thus form conceptions of what we read. In case of lesion of Broca's centre this internal language becomes impossible and the text before the eye of the patient has, accordingly, no meaning for him; a very important source of forming new conceptions is thus cut off. Another very important source is given in the auditory impressions which we receive. The patient with a lesion of Broca's centre can understand what is said to him, but he has lost the power of inwardly repeating what was said and is thus robbed of an important means for establishing landmarks of what he heard, that is, of fastening his attention upon the salient points, and thus forming abstracts of the contents of heard language. In short, the motor-speech centre forms such an important factor in the evolution of the higher mental processes that its lesion cannot remain without damaging influence upon the mental activity. As it also has exquisitely the function of co-ordinating and fusing sensory memories (for instance, visual in reading, auditory in writing from dictation) with memories of motor innervation (for instance, reading, writing) we can understand its location in the frontal lobe. We see at the same time that the physiology and psychology of Broca's centre, as related, corroborates Bianchi's theory regarding the function of the frontal lobe to which that centre belongs.

SOME STUDIES IN HEREDITY.¹

By MARTIN W. BARR, M.D.

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Accepting without question that heredity is law—a law verified by accumulated evidence gathered in every department of science that treats of organic life—some examples of the force of this law as the subject of research to alienists and neurologists may prove interesting to the student and also to the general reader.

Richarz arrived at the following conclusions based upon an exhaustive study of hereditary transmission: First, that mental defect is more frequently transmitted by the mother than by the father. Second, that the inheritance is more likely to fall either to one of the same sex as the parent affected or to the child most strongly resembling that parent. He gives, furthermore, a sequence of liability in inheritance, a liability naturally increased where both parents are neurotic and intensified by consanguineous marriages.

Mother insane, 1. The daughter resembling the mother.

“ “ 2. The son resembling the mother.

“ “ 3. The daughter resembling the father.

“ “ 4. The son resembling the father.

Father insane, 5. The son resembling the father.

“ “ 6. The daughter resembling the father.

“ “ 7. The daughter resembling the mother.

“ “ 8. The son resembling the mother.

Much stress has been laid upon the transformation of type in hereditary neurosis, which does, indeed, occur, amounting not infrequently to the extreme opposite type; thus, an insane parent may bear an epileptic child, or an epileptic parent a child who is a profound idiot. But in solving this, as in many another problem, the difficulty arises from confounding cause and effect. It must be recognized that it is not necessarily a specific neurosis that is

¹Read at the Association of Medical Officers of American Institutes for Idiotic and Feeble-minded Persons, Grand Rapids, Mich., June 10th, 1896.

transmitted, but may be such instability or disordered arrangement of nerve tissue as may evidence itself by different types in various generations according to the degree of prepotency in the mingling of the parental elements.

In the transmission of nervous diseases there are several facts to be noted: First, direct transmission of a neurosis patent, such as epilepsy, insanity or idiocy, from parent to child. Second, the transmission of such instability of nerve tissue from both parents as shall unite to establish neurosis in the child. Third, such neurosis appearing in entirely new type or developing one latent for generations, derived, perhaps, from some forgotten ancestor. This reversion showing unmistakable evidence of the transmission through successive generations of latent disease is one of the most mysterious and at the same time one of the strongest proofs of an inexorable law, written, as Darwin expresses it, in invisible ink awaiting but the applied tests to be revealed.

The reappearance of latent ill is most liable during the decade of development. It is then that great hereditary qualities assert themselves to lift the individual, or atavism to drag him down. To each comes the hour when, like one of old, he must struggle for the mastery of good or ill, as we read: "On the threshold of full reproductive life there is always a liability to break down. The organisms which break down in this way are so pronounced by natural law to be unfit to be reproduced or to live—the tyranny of their hereditary has so doomed them." Rather, let us call it Nature's beneficent opportunity to say, "Thus far shalt thou go and no farther," and, by timely death to the individual, to evolve new life for the race.

Another condition shows the neurosis assuming periodic form, or skipping a generation; thus, in a family of apparently healthy stock its members living to advanced age, one member in every third generation becomes insane. Again, we find a record of a man distinguished in church circles, himself exceptionally clever, his father a dipsomaniac, his son insane. Yet another form of periodicity is that of the neurosis developing in each member of a family at the same period of life.

Piorry relates the history of a family in which every member lost his mind at the age of forty.

Prosper Lucas quotes Michaelis as saying that all the male descendants in a noble German family from the time

of their great grandfather became insane at the age of forty.

Esquirol tells of a family in which for three successive generations the males committed suicide in or near their fiftieth year.

Facts these, stranger than fiction, so fully substantiating Paul Groussac's wonderful idealization that it is worth comparison; indeed, we have here the points we have just been considering focussed as by a camera.

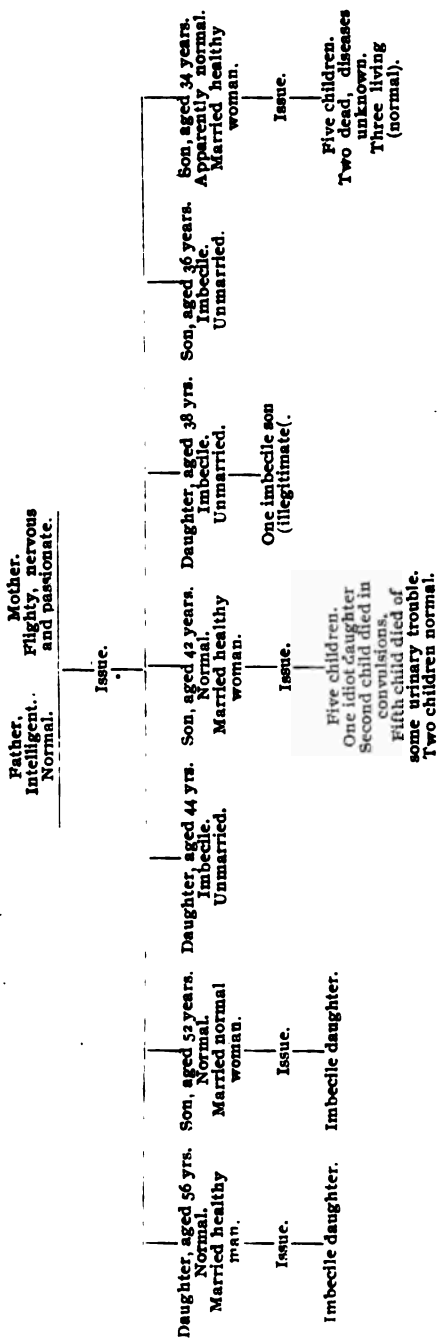
A young Austrian noble, the last of a race whose members for successive generations at the period of fullest development had committed suicide, is kept in profound ignorance of his unfortunate inheritance, and receives every opportunity that new environment and changed conditions of living can offer. An extinct title conferred upon him, educated in England, entering the British navy, he circumnavigates the globe and passes the fateful period in diplomatic service in Washington. There, affianced to the daughter of the minister from B—, he sails with her family for that country. On the eve of landing he seizes his fiancée and leaps from the deck, leaving a letter to his physician and guardian, confessing the horrible impulse that has haunted him for days and to which he knows he must eventually yield.

Clearly associated with this question of hereditary predisposition is that other one of marriage. Burrows, among the first to treat this question as one of grave, practical import, expresses himself "in favor of greater precaution in matrimonial connections," and quotes from Boethius, that in old times when a Scot was affected with any hereditary disease "the sons were emasculated and the daughters banished, and if any female affected by such disease were pregnant she was to be buried alive."

Maudsley inclines to think that to forbid the marriage of a person sprung from an insanely disposed family might be to deprive the world of singular talent or genius, and so be an irreparable injury to the race of man."

Lombroso gives two typical groups: First, the Bach family, presenting, perhaps, the fairest example of healthful mental heredity, which produced through eight generations an uninterrupted succession of musicians, many of high rank, inundating Thuringia, Saxony and Franconia during two centuries. At an annual family gathering are found one hundred and twenty of the name

GENEALOGICAL TABLE No. 1.



of Bach, and among them Fétis counts twenty-nine eminent musicians. Second, the Jukes family, producing in seventy-five years, two hundred thieves and murderers, two hundred and eighty invalids afflicted with blindness, phthisis or idiocy, ninety harlots and three hundred children who died in infancy, and adds:

"In what families can we find genius so fatally and progressively fruitful?"

Morris tells of a distinguished American family in which could be traced harlots through eleven generations, and of another family claiming descent, with bar sinister, from Charles II., whose members inherited the physical beauty, the levity, the gallantry and the fine manners of their royal progenitor.

Moreau, of Tours, said that heredity was found in nine-tenths of his cases, but gives no statistics.

Bucknill and Tuke studied with a view of substantiating hereditary transmission, some six hundred insane families, and out of these found four hundred and forty due to direct inheritance from father or mother, thirteen had insane brothers and sisters and collateral relatives, and one hundred and forty-seven collateral relatives only.

Brigham, out of seventy-nine insane men found forty-two with insane fathers and thirty-five with insane mothers, and in two cases both parents were insane.

Dr. Langdon Down, in studying two thousand cases found forty-five per cent. caused by neurosis in parents.

Ludvig Dahl made careful comparative studies of one hundred and sixty-nine idiots and one hundred and forty-one insane. In the former he found fifty per cent. with insane relations, in the latter thirty-eight per cent. whose relatives presented marked symptoms of idiocy and insanity.

Dr. Kerlin in the analysis of one hundred cases found twenty-eight per cent. caused by hereditary insanity and imbecility and fifty-seven per cent. by neurosis of various kinds.

In my own experience, based upon the careful study of one thousand and forty-four idiots, I find thirty-eight per cent. with a history of hereditary insanity or imbecility and fifty-seven per cent. of other neuroses.

*Vide table No. I.

The following history,² while incomplete in some details, is yet an interesting illustration:

The father, an intelligent man, married a woman who is described as "flighty, nervous and passionate." To them were born seven children—three daughters and four sons.

The first born, a female, normal, married a strong, healthy man and had one child, an imbecile girl. Mother now living, aged fifty-six, in full possession of faculties.

The second, a son, normal, now aged fifty-two, married a healthy woman and has an imbecile daughter; only child.

Third, daughter, imbecile, living, unmarried, aged forty-four.

Fourth, son, normal, now aged forty-two, married healthy woman, but, owing to incompatibility of temper, they were separated. Has had five children, one an idiot girl, one died of convulsions in infancy and another of some kidney trouble. Two surviving children are normal.

Fifth, daughter, aged thirty-eight, imbecile, has had an illegitimate imbecile son, father unknown.

Sixth, male, imbecile, aged thirty-six.

Seventh, male, aged thirty-four, married strong, healthy woman; five children the issue; three living, strong and healthy; two dead, disease unknown.

It will be observed that the mental defect is more pronounced in the female portion of the family.

In the second generation two daughters are imbeciles and but one son, and in the third generation, of the four imbeciles, there are three females and one male. The two normal have each an imbecile daughter, and of the two daughters, the normal has an imbecile daughter and the imbecile an imbecile son.

The next study³ is even more suggestive, including seven generations, therefore giving wider range of observation.

The progenitor, insane, intermarrying with good stock had one daughter, normal, who also contracted marriage with stock, as far as known, pure. Here we find the disease, latent in the second generation, transferred in varied form to every member of the third. The record of her five

²Vide table No. II.

children reads thus: Son and two daughters insane, son and daughter neurotic.

The fourth generation descends through the female line; the youngest daughter a neurotic, having four children; two sons neurotic (pronounced dipsomaniacs), one of whom married a woman with neurotic inheritance; issue, one male child apparently normal. Another son intermarrying with good stock with like result. The only daughter, also normal, married a man of neurotic stock; issue, seven children, apparently normal.

The record of this same fourth generation descending through the eldest daughter, insane but married into good stock, runs thus: Male, neurotic, unprincipled; female, neurotic; female, normal; female, epileptic; female, neurotic; male, normal; female, normal; male, neurotic; male, epileptic; female, normal.

Here, in a generation consisting of fourteen individuals, are six neurotics and two epileptics, with six intermarriages, three partial and three entirely neurotic.

The result shows in the fifth generation about fifty per cent. affected, for of forty-seven individuals there are five insane, one imbecile and seventeen neurotics. Seven of the latter intermarrying with their own kind.

In the sixth generation, we find evidence of Nature's effort to rescue by the power of prepotency where there is admixture of healthy blood; or by barrenness, enfeebled vitality or early death, to arrest neurotic transmission.

The twenty-eight marriages in the fifth generation divide into three groups:

First, normal intermarrying with normal stock.

Second, neurotic with neurotic stock.

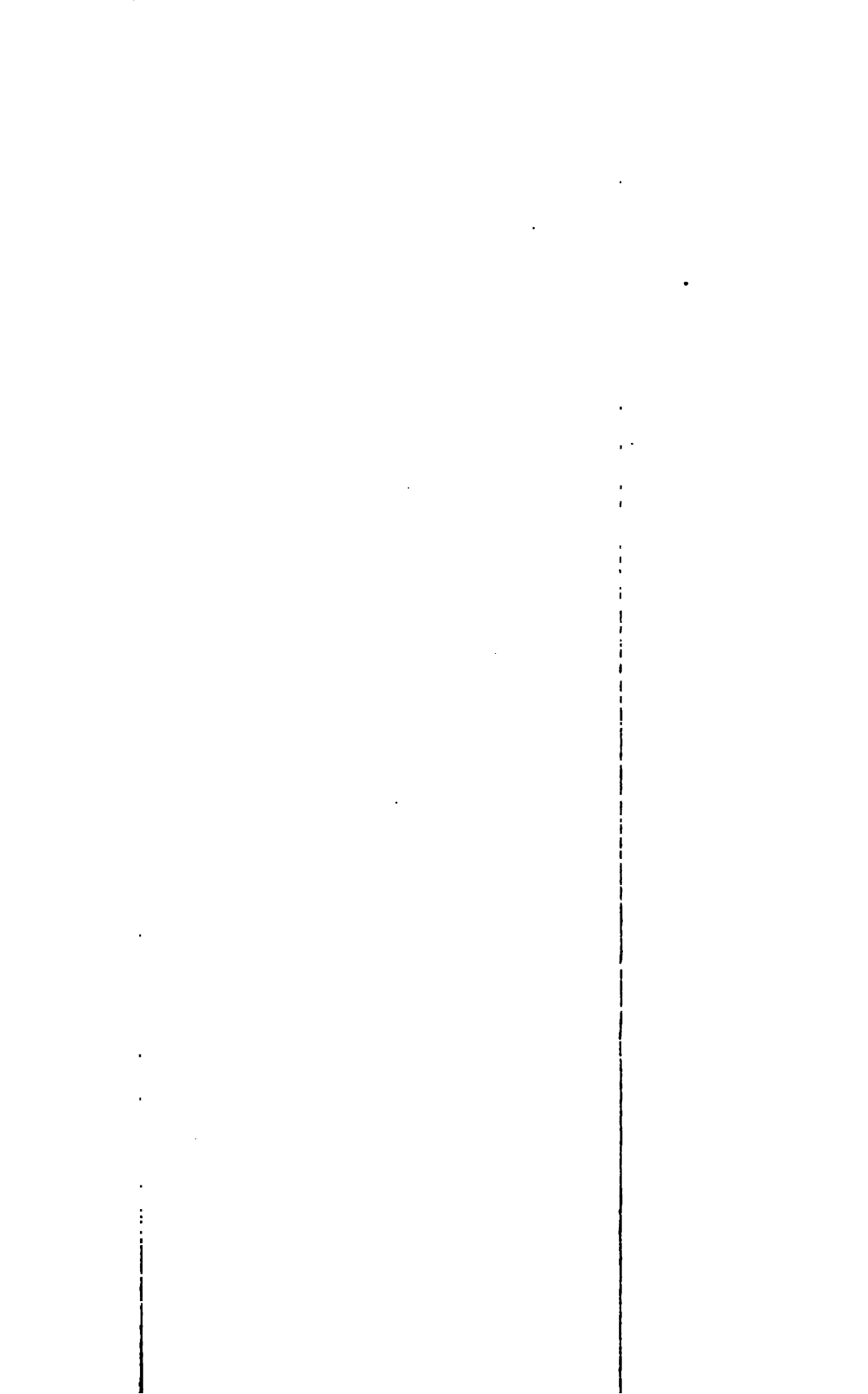
Third, neurotic with normal stock.

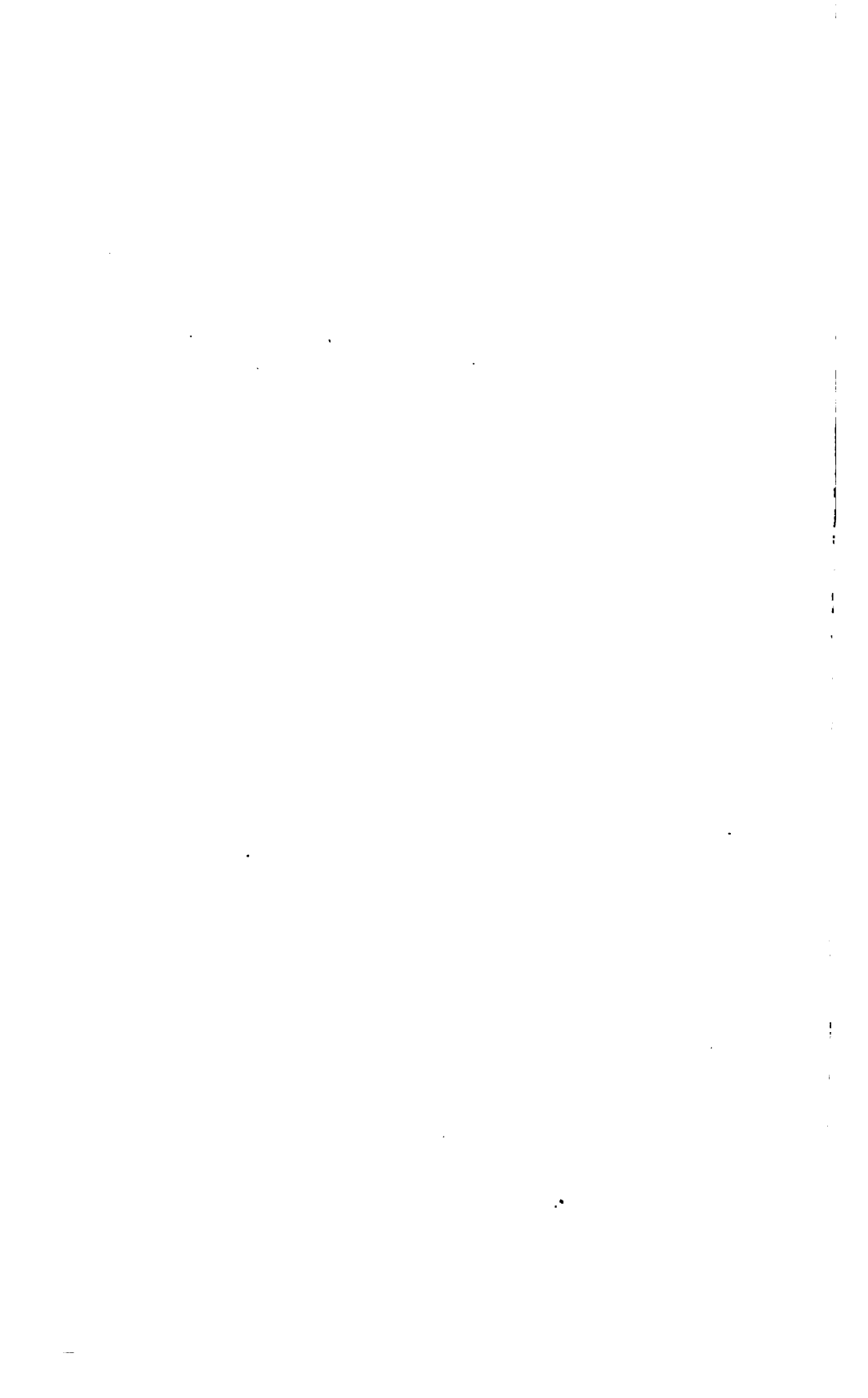
In the first group we find eleven marriages having as issue normal children, but numbering only twenty-two in all; one family of seven, one of four, one of three, one of two, the rest one, and one marriage is barren.

The second group, neurotic with neurotic stock, shows seven marriages with issue of twenty children. Of these, nine are normal, five die in infancy, three are still-born, one is imbecile, one neurotic, one an epileptic, seduced by a negro, giving birth to a mulatto child, making the seventh generation.

The third group, being the union of neurotic with normal stock, shows of ten marriages; ten children normal; one imbecile, one marriage is barren, and of another, that of a dipsomaniac, the two children are still-born.

The entire twenty-eight marriages, with the exception of two, are distinctly not prolific, the prepotency of pure blood asserts itself in the first and third classes, while in the second, early death cuts off nearly one-half the issue, notwithstanding there still remains a sufficient proportion of neurosis patent, not to speak of what we have noted of latent power, to continue the gruesome story and to dominate with evil generations yet unborn.





Clinical Cases.

FIVE CASES OF LOCOMOTOR ATAXIA.

By AUGUSTUS A. ESHNER, M.D.

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Case I.—E. S., a widow, 38 years old, occupied as a seamstress, presented herself at the Howard Hospital with the statement that following an attack of influenza six years previously she had noticed jerking of the legs, principally at night, together with painful sensations. There was subsequently, also, occasional twitching in one or the other shoulder. Vision was subjectively much impaired and the patient from time to time suffered from attacks of vertigo, at times so pronounced as to induce falling. She had, however, never lost consciousness or had a convulsive seizure. She slept poorly and was nervous and easily agitated. The appetite was impaired, the bowels constipated and there was complaint of morning nausea. Headache had been present for four weeks and there had been amenorrhea for four months.

The patient had borne nine children, only four of whom were living, and these were in good health. Several had been born prematurely. The patient's hair was falling out, although she had no knowledge of venereal infection. Her husband had served in the army and had also been a ship-carpenter and a fireman in the city fire department. He died of pulmonary tuberculosis. The woman had suffered from lancinating pains and had some difficulty in walking in the dark, although the feeling conveyed by the floor was natural. There was also some vague girdle-sense.

On examination, the station was found to be swaying and the knee-jerks could not be elicited, even with reinforcement. Coordination was preserved in the upper extremities. The pupils were full, the left being especially large. Both failed to react to light. Dr. W. Campbell Posey, who kindly made an ophthalmoscopic examination, reported the existence of optic atrophy with ataxic move-

ments of the globe. No gross derangement of sensibility could be discovered. The heart presented no lesion.

Case II.—I. K., a married woman, 56 years old, presented herself at the Howard Hospital with swelling of the left lower extremity, which had been present for eight days and had been preceded by pain. Pain was also present elsewhere. The patient further complained of general nervousness. Vision had been impaired for three years and was reduced to light-perception. The symptoms had set in insidiously and their progress had been gradual. Pain had not been an especial or an aggravated feature.

The patient was unable to walk without support and without guidance. She complained of numbness of the feet and inquiry elicited the existence of shooting pains in the legs. There had been girdle-sense during the previous summer, but at no time any crisis. A history of syphilis was not obtained. The woman had borne eight children, three of whom had died, and she had had one miscarriage.

The pupils were full, the left being the larger, but they failed to react to light. Station was unsteady and the knee-jerks could not be elicited. Coordination in the upper extremities also was imperfect. The heart presented no abnormality. The appetite was variable and the bowels constipated. Sleep was fairly good and there was freedom from headache. There was slight vertigo in walking.

These two cases are of interest as occurring in women, as locomotor ataxia is much commoner in men—in the proportion of ten to one, according to Gowers. In neither, moreover, could a syphilitic history or other definite etiologic factor be elicited, although infection with syphilis was strongly probable at least in the first. Finally, in both cases the pupils were large—usually they are small in locomotor ataxia—and by a strange coincidence they were unequal, the left in both instances being the larger. As noted, the pupils presented the reflex immobility typical of posterior spinal sclerosis.

Case III.—J. W., a riveter, 37 years old, was referred to me by Dr. George M. Gould, with the following report of his ocular condition: The fundus presents no lesion. The right pupil is in a state of stabile mydriasis, the left in one of stabile myosis. The external ocular muscles functionate normally. Accommodation is partially paralyzed, parietic.

The patient's parents were living at an advanced age in good health. The father's family was said to be scrofulous. Of seven brothers and sisters one had a disorder whose description corresponded with chorea. The patient's wife had died of pulmonary tuberculosis. Of three children one had died, it was thought, of tuberculosis. There was no knowledge of any miscarriage. While pregnant with the patient his mother had suffered a fall, which apparently left no permanent sequel. The patient himself had had frequent attacks of malarial fever and had also suffered from pleurisy. He had for a year worked with lead, handling pigs of the metal and soldering water-pipes, and had taken no special precaution to prevent absorption of the poison. For many years he had been in the habit of indulging periodically in alcoholic excesses. He used coffee and tobacco freely. He had had gonorrhœa three times, and on one occasion enlargement of the glands in the left groin, with subsequent loss of hair, but without other distinctive signs of syphilis.

The man complained especially of weakness in the lower extremities, which he had noticed for seven or eight years. He had staggered at different times whilst walking when not intoxicated with alcohol. He had also sharp, shooting pains in the legs and thighs, but no girdle-sense. There was no incoordination of the upper extremities, and no conscious difficulty in walking in the dark. The difference in walking upon earth, stone, carpet, wood, etc., was readily appreciated. Micturition was not deranged and the bowels were constipated; there was no incontinence. Sexual desire was diminished and was restrained by a sense of weakness. Vision was blurred and hearing was variable, though impaired. Memory also was impaired. The appetite was good, but food seemed not to nourish or strengthen. There had been no crisis and no loss in weight. At times there was vertigo, with shooting pains in the head.

The right pupil was large, the left small; both failed to react to light, the right also in accommodation. The right palpebral fissure was narrower than the left. The patient had been struck over the right eye some years previously with a bag of hemp weighing about three pounds. Some of the hemp entered the eye and an obstinate inflammation followed. The pupil had remained large since this accident. The gait was rather awkward, especially in turn-

ing about. Station was a little unsteady, and the more so when the eyes were closed. No ataxia could be detected in the upper extremities. The knee-jerks were wanting and were not influenced by reinforcement. General sensibility was preserved. The action of the heart was rhythmic, the sounds clear. There was no blue line appreciable at the margin of the gums.

In this case we have three etiologic possibilities: syphilis, lead and alcohol. Of the first we have no definite evidence, but exposure to the risk of infection makes it impossible to exclude it as an etiologic factor. Of intoxication with lead and alcohol there is no denial. What part each or all of these influences played in the causation of the disease must remain undetermined.

In this case, too, the pupils were unequal, the right being large, the left small. It is probable, however, that the conditions in the right eye were dependent upon local changes and in no way related to disease of the cerebrospinal system.

Case IV.—J. W. L., a widower, 43 years old, was referred to me at the Polyclinic Hospital and related that he had for several years been troubled with bleeding hemorrhoids, which were from time to time a source of pain and annoyance. He had also noticed pain of a shooting character in the lower extremities. His appetite was unimpaired, his bowels regular and he slept well. The tongue was coated. The man had no difficulty in walking, subjectively or objectively, and his station was steady. The pupils, however, were small and immobile on exposure to light, although they reacted in convergence and to accommodation. The eyes were examined carefully by Dr. Jas. Thorington, who reported, in addition to the absence of the pupillary reaction to light-stimulation, that both irides were round, the right measuring two mm., the left one and a half mm. in diameter. Both discs were round, and in the right, just above the macula, there was a small area of low-grade choroiditis. The visual fields were contracted for both form and color, especially for the latter. The knee-jerks could not be elicited, even with reinforcement. There was some weakness of the sphincter of the bladder. There had never been girdle-sense or crisis of any sort. Sensibility was preserved and the coordination of the upper extremities was good. The heart presented no lesion. The patient complained of

slight pain on manipulation of the bare back. He had had an attack of enteric fever at the age of nineteen. He indulged in alcohol and in tobacco freely, but denied absolutely any venereal disease. For a time he had lived on the Western plains and had camped out and roughed it a good deal.

In this case we have only the abolition of knee-jerks and of pupillary reflex, together with lightning-pains and weakness of the vesical sphincter, upon which to base a diagnosis of posterior spinal sclerosis, and, in the absence of other evidence to the contrary, these may be considered sufficient. Literally, the case is not one of locomotor ataxia, that is, at the present stage, there being no apparent disorder of locomotion or ataxia of any kind. There can be no doubt, however, of its being one of posterior spinal sclerosis, and it is not unreasonable to assume that the motor disability will appear in the further progress of the disease. This is not a rare sequence of events, and the incoordination may be deferred for a long time and even fail entirely to appear. Lightning-pains and loss of knee-jerks are usually the earliest symptoms of the disease.

The origin of the disease in this case may be attributed either to a life of exposure or to alcoholic excess, perhaps to the conjunction of both influences.

Case V.¹—E. A. R., a married man, 49 years old, employed as a night clerk in an express office, presented himself at the Howard Hospital on account of attacks of vertigo of which he had had four in the preceding six months. A year before he had had an epileptiform convulsion, in which he had lost consciousness and had bitten his tongue. This attack had occurred at 5 A. M., before the patient had arisen from bed and a day or two after hearty indulgence in crout. There was no paralysis. He complained of formication of the left forearm and wrist, associated with a shock-like sensation along the ulnar aspect of the hand. In the attacks of vertigo the patient became blinded and reeled and he thought he would fall if not supported. Subsequently, he manifested a tendency to turn toward

¹This patient was exhibited by Dr. Chas. K. Mills at a meeting of the Philadelphia Neurological Society, held Oct. 23, 1893, and an account of the case appears in *The Journal of and Nervous Mental Disease*, 1894, p. 127. It was then considered as a probable case of posterior sclerosis of slow development. At that time, however, no changes were found in the fundus.

the left. On beginning to eat, tears would appear in his left eye and the left side of the face would break out in profuse perspiration. Memory was deranged, so that the patient could recall facts but not names. There were no delusions of exaggeration. Face, head and lips were tremulous. The man stated that at times he trembled in writing, though in my presence he was able to write legibly, though a little jerkily. This character of writing, he said, was customary in his occupation. The gait was a little clumsy and halting and not made much worse by closing the eyes. In walking there was a tendency to deviate toward the left. The patient was able to stand steadily on both feet, with eyes open or closed; less steadily on either foot alone; and least of all on the left foot. The left pupil was the larger. Both were fairly regular and reacted in accommodation but not to light-stimulation. The tongue was tremulous and appeared to deviate slightly toward the right when protruded. The sense of taste was preserved, that of hearing was defective and worse upon the left side. The tick of a watch could be heard at a short distance from the right ear, but only when in contact with the left. A feeble, though at times distinct, knee-jerk could be occasionally elicited on the left and it could be reinforced; none at all, however, could be discovered on the right, though at times it was thought a feeble contraction of the rectus femoris could be felt and slight extension of the leg seemed to take place. Common sensibility was grossly preserved, although there appeared to be some blunting in places. The reflexes in the upper extremities were preserved. There had never been girdle-sense and the sense of feeling in walking was normal. The sphincters were under perfect control. Sexual desire had been wanting for a year. The appetite was preserved and the bowels constipated. The patient's countenance was mobile, and he stated, on inquiry, that he was not unduly emotional. The grasp of the hands was quite strong. After taking potassium iodid, gr. x, and mercuric chlorid, gr. 1-24, for a day, the patient noted profuse lachrymation in both eyes, with aching of the teeth and jaw-bones and also of the eyes. After a purge and a mustard foot-bath the right eye became completely closed. On the following day the left eye became closed, while the right was again open.

It was learned further that on July 4, 1876, the patient had been overcome by the heat and that his pupils were dilated at the time. He had had an attack of gonorrhœa, but denied other venereal infection, and there had been no obvious secondary manifestations of syphilis. His wife had borne five healthy children and had suffered no miscarriage. The patient had at different times taken alcohol to excess and he chewed tobacco freely. He was subject frequently to attacks of sneezing, lasting several minutes at a time. No neuropathic heredity could be elicited. The urine contained no albumin. Speech was a little thick. Examination of the eyes by Dr. W. Campbell Posey revealed: Argyll-Robertson pupil on both sides, with ataxic movements of the globes at the extreme limit of rotation. Optic neuritis was present in both eyes, passing into the atrophic stage, and more marked on the right side. The fields of vision, for form and for color, were contracted concentrically. After taking picROTOXINI, gr. 1-30 t. d., the patient said that he felt better, although he complained of a shock-like sensation upon the ulnar aspect of the left hand. There was no perspiration upon the right side of the body. The knee-jerks could not be elicited at a second examination, when also station was rather unsteady. There had been no nausea or vomiting and no headache. There was generally no spontaneous vertigo, but dizziness was induced by stooping.

The nature of this case is not entirely clear. There are present many symptoms of posterior spinal sclerosis: the awkwardness of gait, the uncertainty of station, the failure of the pupils to react to light, the optic-nerve degeneration, the hypesthesia, the enfeeblement of the knee-jerks. Besides, we have some of the symptoms of general paresis: the tremulousness, the thickness of speech, the impairment of memory, the epileptiform convulsions. There remain yet to be accounted for, however, the vertigo, with deviation to one side, the impairment of hearing, the vasomotor manifestations and the preponderance of some of the symptoms upon the left side. These are suggestive of a basal lesion, and with a history of insolation it is not difficult to go a step farther and attribute the manifestations in part at least to a meningitis involving pons, medulla and cerebellum. In reply to an inquiry whether the ocular findings best adapted themselves to a diagnosis of locomotor ataxia (posterior spinal sclerosis), of general paresis

or of chronic or old basal meningitis, Dr. Posey stated that "there is nothing in the ocular findings which would prohibit, in fact, the findings would rather incline to, a diagnosis of chronic or old basal meningitis." While the diagnosis is, perhaps, not entirely adequate or satisfactory, it seems best to account for the varied symptoms present. It is not impossible, of course, that there may be two sets of lesions—a posterior sclerosis in conjunction with a basal meningitis. In this case the pupils, while small, were, like the pupils in Cases I., II., III. and IV., unequal, and again it happens that, as in Cases I. and II., the left was larger than the right.

Cases I. II. and V. were seen in the service of Dr. Lewis Brinton at Howard Hospital, and it is to his courtesy that I owe the privilege of reporting them. I desire to thank Dr. Posey for his kindness in furnishing careful eye-reports in Cases I. and V., and Dr. Thorington for similar report in Case IV.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, Dec. 1, 1896. B. Sachs, M.D., President.

CONGENITAL DEFECTIVE OCULAR MOVEMENTS.

Dr. W. Leszynsky presented a boy of four years who had been referred to him on April 21, 1896, by Dr. A. B. Tully. The child had been born at full term, after a short and normal labor. Between the fourth and fifth months of pregnancy, the mother had been greatly frightened by seeing a child killed by a fall. Toward the end of the first year, the child began to draw his head backward, and it was then noticed that his eyes turned inward. Examination last April showed convergent strabismus, pupils normal; tongue protruded well; teeth well developed. The measurements of the head came within the physiological variations of the fully developed adult skull. The heart and lungs were normal. Dr. Tully found a hypermetropia of four dioptries. Glasses were fitted to the eyes, and on his next visit the backward movement of the head had much diminished, as had also the convergence of the eyes. There was slight lateral oscillation of the right eyeball. He was unable to move either eye outward beyond the middle line. There was absolutely no action of either externus, showing that conjugate action was not wholly absent, being performed by the respective interni. There was no indication of abnormality of the other cranial nerves. The parents of this child were healthy, and there were six children, all healthy and well developed, mentally and physically. The speaker said that uncomplicated congenital absence of outward movement of both eyes, so far as he knew, had been quite rarely observed. All recent writers agreed that the terms, "abducens paralysis" and "nuclear paralysis" were inaccurate. These congenital defects were almost always due to defects in the development of the ocular muscles, and the nerves were usually normal. Post-mortem examination had shown complete absence of the muscles, or if they were present, they were ill-developed, or attached to the eyeball in abnormal positions. The case presented was one of congenital arrest of development, affecting the recti muscles, or possibly, a case of faulty insertion of these muscles.

Dr. Terriberry asked if the pharyngeal vault of this child had been examined for adenoids, as the position of the head and the expression of the face would suggest the presence of such a condition.

Dr. Leszynsky replied that this had not been done, but the position of the head seemed to be due to the effort made to see objects above the ordinary plane of vision.

The President said that he had reported one case of double ptosis, with double rectus externus paralysis. He had always regarded these cases as examples of congenital nuclear defect. It seemed to him rather difficult to uphold the view that it was a muscular defect.

HYSTERICAL AMBLYOPIA.

Dr. J. Arthur Booth presented a case of functional or hysterical amblyopia. The patient, a young woman, had had an attack of la grippe two years ago. Previous to this, her health had been good, but since then she had not been well. There had not been any trouble with the eyes, however, until about two weeks ago, at which time she suddenly became blind in one eye. When he saw her, three days ago, there was an entire absence of any lesion of this eye. He proposed to try treatment by hypnotism, and would present her at the next meeting of the Society to show the result of this treatment. He had given her one séance, and as a result, she had been able to see the fingers at a distance of about two inches. She is anæmic, has crying spells, and a globus hystericus.

MUSCULAR FATIGUE FROM OCCUPATION.

Dr. Terribery presented a male negro, thirty-two years of age, who had been a cigar maker for the last twenty years. With the exception of malaria in his youth, he had not been sick, and had been temperate in the use of both alcohol and tobacco. There was no evidence of syphilis. In the latter part of August, 1895, he noticed a slight weakness in the right hand. This became so pronounced soon afterward that he was compelled to quit work for a few months. Examination of the right upper extremity showed it to be entirely normal, with the exception of slight weakness in the *general* movements. The position of the affected hand was also somewhat different from that of the other. There were no sensory phenomena, and the voluntary motor paths were free. The speaker said that he presented the case chiefly to exhibit the peculiar attitude assumed when the man endeavored to get through the motions of rolling a cigar. After the first movement, it would be observed that the hand rolled out into complete supination. There did not appear to be any spasm of the muscles involved, but the hand was supinated by the preponderating action of the unaffected muscles.

SYRINGOMYELIA WITH PECULIAR SENSORY SYMPTOMS.

Dr. Hirsch presented a youth who had come to him some time ago complaining only of pain in the left arm. Examination showed very marked atrophy of the ulnar region, and the hand in the usual position indicative of ulnar paralysis. In addition to this there was atrophy of the supra- and infra-spinatus muscles. The left pupil and the left fissure of the eyelid were smaller than those on the other side. The left patellar reflex was exaggerated. In several regions of the body there was marked analgesia with loss of the power to distinguish heat and cold. The patient said that the condition of the eyes had existed from early childhood. It was possible, therefore, that a gliosis or some other lesion had existed since childhood, so that at first the cilio-spinal centre was affected. Examination of the eyeballs showed intentional nystagmus. If he looked to the right there would be a typical rotatory nystagmus, while it would be a purely lateral nystagmus if he looked to the left. Another peculiar feature was the distribution of the sensory disturbances. There was an area of marked analgesia extending down to the fifth rib, and over both arms, while there was even hyperæsthesia in both palms. There was also analgesia on the right half of the face, but not up to the median line. There was also a region of hyperæsthesia around the hip. In the regions showing analgesia he could not appreciate heat or cold. On the other hand, while the palms were hyperæsthetic, he could not distinguish here the difference between heat and cold. Another peculiar feature was that if the analgesia were tested centripetally, the border of the area would be removed toward the centre, whereas if tested centrifugally, the border of this area would be removed from the centre. The extent of this movement in the direction of the test was a little over one inch.

ENCEPHALITIS FOLLOWING SUNSTROKE.

Dr. J. Winters Brannan presented a woman, forty-two years of age, a laundress, who had been brought to Bellevue Hospital on August 10, 1896, suffering from sunstroke. She was unconscious on admission, and had a temperature of 110° F. She had been in good health previously, but had been somewhat intemperate. Under the use of ice baths and stimulants she rallied from the sunstroke, although as late as the fourth day the temperature was 105° F. She remained in bed for about one month, and at the end of two months was able to walk a little with assistance. Her speech had remained quite indistinct. The movements of the arms were tremulous and decidedly incoördinate. Sensation was preserved everywhere.

The knee reflexes are almost entirely lost; there is no ankle clonus. Reaction to electricity is normal above the knees. There is no reaction to a strong faradic current below this point. Dr. Weeks examined the eyes with negative result. On attempting to walk, she nods her head. He looked upon the case as possibly one of multiple sclerosis.

Dr. C. L. Dana said that he had seen a somewhat similar case following sunstroke, and he had looked upon it as one of non-suppurative encephalitis. Of course, encephalitis in this instance had subsided, and had left sclerotic patches in the cortex, and probably also in the base of the brain. She had had a high temperature, and necessarily, therefore, such cerebral congestion, that it was natural to suppose that there were a number of points of capillary hemorrhage with an inflammatory reaction leaving scars in the cerebral tissue. The case just presented seemed to be an exaggerated type of a class which were not particularly rare after sunstroke. It might be complicated by a neuritis, due to her alcoholic habits, which would explain the condition of the knee-jerks and the unsteadiness.

Dr. Brannan said that the pupillary reactions were normal, and that it had been suggested to him at one time that she was suffering from a multiple neuritis.

The President said that during the past summer he had seen a number of cases in private practice after the period of severe heat, in which there were distinct symptoms of a general encephalitis. Some of the cases presented all the symptoms of a "convexity encephalitis," and one or two of them presented in addition distinct bulbar symptoms. Possibly the difficulties of speech in the case just presented might be interpreted as being in part due to an involvement of a basilar portion of the brain. The diagnosis of multiple sclerosis could hardly be accepted, as the symptoms were not sufficiently pronounced.

MULTIPLE SCLEROSIS IN CHILDREN, WITH A REPORT OF THREE CASES.

Dr. L. Stieglitz read a paper with this title. He stated that the first reported case of multiple sclerosis in children had been published in 1881, together with the post-mortem findings. He had had the good fortune to observe three cases of the disease in children during the past year. A study of the literature of the subject had forced him to believe that the diagnosis of multiple sclerosis in a number of the reported cases could fairly be challenged.

His first case was a girl of nine years, who had been born after a somewhat prolonged labor. The parents were healthy,

and there was no history of syphilis in any member of the family. When three months old, a number of furuncles appeared on the face and neck, and starting from one of these, an attack of erysipelas developed. There was prolonged high fever, but no convulsions. Soon after this it was noticed that her hands trembled. Her speech had always been slow. Two years ago she had a severe attack of scarlatina, and for some time after this the speech was worse. She is more or less emotional. When first seen on October 17, 1896, the pupils were equal and responsive, and there was no nystagmus. There was slight tremor of the tongue; the deep reflexes of the arm were lively, and the knee-jerks were exaggerated; ankle clonus could be elicited in the right foot. There was a marked intention tremor in both arms; there was no muscular atrophy. Her voice was harsh, and the speech slow and monotonous. Mentally she was backward.

The second case was that of a girl of eleven years, who had been seen first in September, 1895. There was no history of previous nervous or mental disease in the family. Her eyes had always been very prominent. For the past year she had lost control of the bladder very largely. She had also been weak, and unsteady on her feet. She was fairly developed; there was divergent squint and a fine nystagmus. The ophthalmoscope showed optic atrophy in both eyes. The pupils were large and responsive. There was a fine tremor of the fingers when at rest, and a marked intention tremor in both hands. The gait was that of an ataxic paraplegia of cerebellar origin. The knee-jerks were greatly exaggerated, and the ankle clonus was present on the left side. Sensation was perfect in all parts of the body. The speech was not affected, and the child appeared to be quite intelligent. Ataxic paraplegia, the speaker said, could be excluded by the eye symptoms.

The third case was that of a girl of fifteen years, first seen on March 30, 1896. Shortly after an attack of what was apparently influenza, walking became unsteady and speech difficult. Since then she had been subject to slight vertigo. Her voice was harsh, and speech required an effort. There was no nystagmus, and the pupils were responsive. The deep reflexes of the arms were very lively. The gait was that of spastic paraplegia. The knee-jerks were greatly exaggerated, and there was ankle clonus on both sides. She possessed average intelligence.

Regarding the differential diagnosis, the speaker said that the following diseases would require consideration: Infantile cerebral palsy, syphilis of the cerebro-spinal axis, hereditary cerebellar ataxia, acute disseminated myelitis, tumor of the brain and hysteria. It was often very difficult to distinguish between infantile cerebral palsy and multiple sclerosis. In the

large majority of cases of the former the question of the presence of disseminated sclerosis would not arise at all. Infantile cerebral palsy is either congenital or acute in its onset, and these facts are of much importance in making the diagnosis. Although the morbid process might not be absolutely stationary, it was always localized and could not affect distant parts of the cerebro-spinal axis. In disseminated sclerosis the onset might be either acute or sub-acute, and the morbid process break out fresh again and again, adding new symptoms to the old. In a word, it might be said that multiple sclerosis is progressive and retrogressive; infantile palsy is stationary. Intention tremor, nystagmus and bradyphasia are the rule in disseminated sclerosis, and the exception in infantile cerebral palsy. Syphilis of the cerebro-spinal axis, he said, was rare, but such cases had been reported in young children. The same considerations which hold good in the adult were of value in the young. Friedreich's ataxia was to be distinguished from multiple sclerosis by the knee-jerks being lost or diminished, the pupils normal, the speech halting and explosive, and by the rare occurrence of ocular nerve palsies. In hereditary cerebellar ataxia the speech was hesitating and abrupt; there was a jerking nystagmus with optic atrophy and a contracted field of vision. Absence of heredity was of no diagnostic value. The presence of ankle clonus, nystagmus or optic atrophy would at once exclude hysteria.

Regarding the etiology of the disease, Dr. Stieglitz said that both neuropathic taint and infective diseases seemed to play a part. The prognosis was more favorable in children than in adults.

THE RELATION OF MULTIPLE SCLEROSIS TO MULTIPLE CERE- BRO-SPINAL SYPHILIS.

Dr. B. Sachs read a paper on this subject. He said that between multiple sclerosis and syphilitic diseases of the brain and cord there was an exceedingly close resemblance. In multiple sclerosis and cerebro-spinal syphilis both the brain and cord might be affected. Both occurred between the ages of twenty-five and forty years. Nystagmus, intention tremor and scanning speech might be placed to the account of multiple sclerosis, yet he had seen nystagmus and scanning speech in some cases of cerebro-spinal syphilis. Syphilis was not considered by many authors to be a factor in multiple sclerosis. He detailed several cases illustrative of the difficulties experienced in making the differential diagnosis. If the disease invades the brain, the presence of nystagmus and scanning speech would help to determine the true nature of the disease,

but not so if it invaded the spinal cord. He thought the greatest stress should be laid upon the ocular conditions. In syphilis of the brain, nystagmus is rare. Ocular palsies occur in both affections, but are rarely as complete in multiple sclerosis as in syphilis of the brain. It was rare to find ocular palsies precede the development of other symptoms by a year or more as they often did in cerebral syphilis. He felt convinced that a *complete* immobility of the pupils on exposure to light and during accommodation is more common in syphilitic affections than in any others. This condition of the pupil could be observed also in those forms of hemiplegia due to a specific endarteritis. A difference in the manner of response of the pupils was also very common in syphilitic affections. If all other signs fail, the behavior of the pupils would be a strong evidence in favor of syphilis rather than of multiple sclerosis. The complete immobility of the pupil is often the earliest and the most persistent symptom in brain syphilis. The optic nerve might be involved in both multiple sclerosis and brain syphilis. Optic neuritis was more common in syphilitic affections, while a partial atrophy was much more characteristic of multiple sclerosis. The very complete remission of all the symptoms for a period of months or years was very characteristic of brain syphilis. It was not impossible to conceive of sclerotic patches developing in persons previously infected with syphilis. The question could only be finally settled by careful investigation of the blood vessels.

Dr. C. L. Dana thought that due credit should be given to Dr. Fränkel for having made the diagnosis of multiple sclerosis in one of the cases before even Dr. Sachs or himself. He could not agree with Dr. Sachs that the other case was not one of general paresis.

Dr. Joseph Fränkel said that in one of the cases reported in Dr. Sachs' paper, that there was an intention tremor of the head. It was present only when he turned his head in a certain way, and in the beginning of the disease it was not always to be made out. The cephalic extremity rarely manifested "intention" tremor, but when it did, the tremor was no less characteristic than when of other parts of the body. Regarding the pure type of multiple sclerosis, he said that he would lay more stress upon the psychical symptoms than is usually done. Patients with this disease have a peculiar expression of the face, the face being drawn up in a spastic state, and this spastic condition of the face is a manifestation akin to that existing in the extremities. There was a spastic condition in the lower extremities—a resistance to passive movements. In these patients we get a clonus of laughter or a clonus of crying, just as in the lower extremities we get an ankle clonus. These peculiar psychical features are not observed in cerebral syphilis.

There was a higher state of spasticity in multiple sclerosis than in cases of specific paraplegia. This furnished a point of differentiation in some cases of hysteria. The lack of proportion between the spasticity and the amount of motor paralysis would enable one in some instances to make the differential diagnosis.

Dr. Hirsch said regarding the differential diagnosis of multiple sclerosis and general paresis, that he would like to ask how Dr. Sachs could distinguish multiple sclerosis from general paresis by the psychical symptoms. He knew of no such psychical symptoms. Nor could he understand how the diagnosis of multiple sclerosis could be made from the behavior and the expression. These symptoms were very well marked in general paresis. From the simple dementia up to the delusions of grandeur everything might be developed in general paresis that could be offered in psycho-pathology. The remissions referred to by Dr. Sachs would by no means exclude general paresis. While in many cases it was impossible to make the differential diagnosis between these diseases, the course of the disease was of some assistance. Multiple sclerosis, as a rule, lasted longer than general paresis. The inequality of the pupils, or of the two halves of the face, would be in favor of general paresis rather than of multiple sclerosis.

Dr. Edward D. Fisher said regarding this matter of diagnosis by the psychical symptoms, that in cases of multiple sclerosis there was often a blankness of expression when the face was at rest. The laughing and crying of these persons were often observed, but this was not a psychical state, because they did not while laughing or crying experience either joy or grief.

Dr. Pritchard said that he had been interested chiefly in the form of the disease as observed in childhood. He had been at first much surprised to find that the disease was as common as was indicated by the number of reported cases, but on careful analysis a number of these cases had had to be excluded. There were still, however, 50 or 60 well authenticated cases on record. As to the relationship between cerebro-spinal syphilis and multiple sclerosis, there should be very little occasion for confusion in children. In differentiating in adults, the insomnia, the nocturnal headache and the exacerbations of the one disease, were in marked contrast with the entire absence of periodicity or characteristic headache, or insomnia observed in cases of cerebro-spinal sclerosis. He thought Dr. Stieglitz had not laid sufficient stress on the differentiation of cerebro-spinal sclerosis from certain of the anomalous forms of so-called chorea. In a great many cases of supposed infantile cerebral palsy there was a condition corresponding exactly to the disease known as multiple cerebro-spinal sclerosis.

Dr. Stieglitz said that he had simply included in his paper those diseases which were represented in the literature by cases reported as multiple sclerosis, and in which, he thought, there was reason to believe the diagnosis might have been different.

Dr. Sachs, in closing the discussion, said that it was for the purpose of bringing out the resemblance between multiple sclerosis and the forms closely related to it that this discussion had been planned. The case referred to in the paper in which Dr. Fränkel had made the diagnosis of multiple sclerosis upon one symptom—the tremor—was instructive, as neither Dr. Dana nor himself had been able to make this diagnosis as they had not been able to see the patient when this tremor was present. He was positive that the case described in his paper was not one of general paresis, as some of the speakers seemed to think. The pupillary symptoms had remained practically normal from the beginning to the end, and the complete spastic paraplegia from 1889 to 1892. There had not been a particle of mental disturbance. The only differential diagnosis, it seemed to him, was between multiple sclerosis and cerebro-spinal syphilis. He could not allow that this was an ordinary case of general paresis. The difficulties of diagnosis were well exemplified in children. He had also seen the second case reported by Dr. Stieglitz, and at that time had been undecided as to whether the case was one of cerebellar ataxia or multiple sclerosis. He still thought that the optic atrophy and marked cerebellar gait were in keeping with the diagnosis of cerebellar ataxia. It was only on account of the presence of the peculiar tremor that he hesitated about the diagnosis.

PHILADELPHIA NEUROLOGICAL SOCIETY.

October 26th, 1896. President, Dr. Charles K. Mills, in the chair.

Drs. DeForest Willard and Wm. G. Spiller read a paper, entitled: Concussion of the Spinal Cord ("Railway Spine").

ABSTRACT.

A case of fracture of the spinal column at the eleventh thoracic vertebra was described. After the accident there had been complete paralysis of motion and of sensation below Poupart's ligament, except on the outer part of the thighs. Although the spinal cord was much injured at the level of the first to the third lumbar segments, and to a less degree above this portion, sensation had been preserved in the areas innervated by the external cutaneous nerves. It has been shown experimentally that it is necessary to cut at least three spinal roots in order to destroy sensation in any given region. Probably the preservation of sensation on the outer part of the thighs was due to the presence of nerve filaments from higher levels than the first lumbar segment. The patient died five days after the accident, chiefly from exhaustion.

At the autopsy the dura was found intact, and no hemorrhage was observed within. The cord, even at the seat of fracture, was quite firm and of normal shape, and presented no indications externally of injury from pressure. The microscopical findings consisted of displaced fibres in one portion of the cord, numerous hemorrhages, altered blood pigment, masses of granular corpuscles, necrosed tissue, greatly swollen axis cylinders, degenerating myeline sheaths, tumefied ganglion cells, and some round cell infiltration. Some of the axis cylinders of the spinal roots were swollen, and the myelin did not stain as deeply as in normal sections. The case was very similar to the one recently described by Westphal, and while neither could be called a case of pure concussion, many of the findings were evidently the result of this condition. Westphal states that in most of the reported cases of traumatic myelitis death has occurred either immediately or else a long time after the accident, and that these are unfitted for a study of the early morbid changes.

Drs. Willard and Spiller refer to the many cases of "traumatic back" which are too often regarded merely as evidences of neurasthenia. Such lesions as they describe are doubtless present in less degree in many of these patients, though frequently associated with symptoms of neurasthenia. The restoration of function does not exclude the presence of organic changes, as shown by the remarkable case of Pott's disease reported by Charcot. They claim that they are not advancing an ungrounded theory. Obersteiner, Schmaus, Bikeles, Struppler, etc., have proven beyond question that organic changes in the spinal cord may follow trauma without fracture.

DISCUSSION.

Dr. F. X. Dercum.—I think we are much indebted to Drs. Willard and Spiller for this interesting contribution. We will probably all admit that we have in the past erred in calling some of the cases of "railway spine" functional. We should remember, also, that the fact that recovery sometimes follows after a time does not prove that organic changes in the cord have not been present. Again, it is not difficult to recall cases regarded as functional in which recovery has never ensued. I myself have frequently erred, I think, in laying too much stress upon the mere strain of the muscles and fibrous tissues of the back, and of laying too little weight on the motor and sensory disturbances as indicating probable changes in the cord.

As our knowledge progresses there will probably be left no functional diseases whatever, and lesions of the neurons will be demonstrated from almost all of the symptoms with which we meet.

Dr. G. Betton Massey.—This is an interesting subject and has an important medico-legal bearing. We are all apt to look with suspicion on patients who are suing companies, and to make the mistake of regarding symptoms as due to neurasthenia at least, if not to malingering.

Dr. Dercum and I recently saw a gentleman who two years previously had been injured in a railroad accident and had undergone considerable immediate treatment in a hospital. At the time I saw him, it was easy to believe that the man was a malingerer, if you were at all suspicious. That man had led an active life, so far as the upper portion of the body was concerned, during the year previous to the time I saw him, and yet there was apparently functional paralysis of locomotion. The nutrition was pretty good. I allude to the case only to call attention to the electrical tests which proved that the plaintiff had had a considerable injury of the cord from which he had not recovered. The examination proved that the man was and had been a serious sufferer.

Dr. Chas. W. Burr.—It seems to me that the clinical history of this case is not the kind that could properly be called one of "railway spine." The man was suddenly paralyzed from the waist down, with complete paralysis of the bladder and rectum, and had a fracture of the spine itself. In ordinary "railway spine" the symptoms come on more slowly and gradually get worse. I think myself that many of the cases considered to be functional, are really due to organic changes. In Drs. Willard and Spiller's case, I think that we could have said with certainty that the spinal cord was seriously injured, without making a post-mortem examination.

Dr. F. X. Dercum.—The distinction which Dr. Burr makes is, of course, important. Practically, we should distinguish between local concussion of the spine and that condition of general nervous shock which is understood by the expression "railway spine."

Dr. Chas. K. Mills.—While this was not a case of "railway spine" in the ordinary acceptance of the term, I understand that the intention of the writers is to teach us something of what happens in the cases known as concussion of the spine, by what occurs in the more severe cases of this character. In this case there was fracture and hemorrhage, but in addition there were found in various parts of the spinal axis evidences of injuries which were not directly connected with the gross spinal lesions. From this point of view the case is important, because we cannot expect to get autopsies in cases of the usual type. I have long been convinced that, in at least some cases, gross lesions occur. I believe that they are in the nature of small hemorrhages, particularly in the gray matter of the cord. Ever since the experiments of Duret on cerebral concussion, I have thought that an explanation similar to that given by him might apply to spinal concussion, although not so fully on account of mechanical and other differences.

Long ago I published in the *Medical Times* a case of intracerebral hemorrhage with inundation of the ventricles, which bears upon this subject. When you have intracerebral hemorrhage you have the brain struck a blow from within, just as in external traumatism you have it struck from without. In these cases there are many lesions. In addition to the gross hemorrhage there are in various parts of the brain ecchymotic areas and also minute hemorrhages in the white matter—capillary or small arterial extravasations. It seems to me that similar conditions must be of frequent occurrence in the spinal cord, and just as in brain cases many patients recover in spite of a number of minute lesions, so in the cord cases, recovery not infrequently ensues.

I have often wished that some one would throw more light on those cases of persistent anæsthesia which are sometimes

seen. There is some inkling of light in the fact of the greater tendency to the occurrence of lesions of a certain kind in the posterior regions of the cord.

Dr. Spiller in closing the discussion said: It was not our intention to call this a case of "railway spine." The diagnosis of fracture of the vertebral column was made. Certain of the lesions as displacement of fibres, were due to purely mechanical causes; there were others, however, which could not be attributed to the fracture, but were the result of the blow which was severe enough to cause the fracture. Similar lesions have been found after concussion in cords when fracture has not occurred. It was, therefore, considered proper to attribute certain of the lesions in our case to concussion. A nerve fibre which has been injured may for a time be capable of function until gradually increasing degeneration destroys its usefulness. We may in this way, perhaps, explain the development of symptoms in "railway spine."

Dr. Mills has referred to lesions in the posterior horns.

It has been shown experimentally (Goldscheider and Flatau) that a colored fluid injected into the anterior horn has a tendency to pass to the posterior horn, and to ascend in this, if injected into the posterior horn, it does not enter the anterior to any extent. Blood poured out into the cord probably takes the same course.

We do not wish to go on record as denying the functional nature of many of these cases. We simply claim that frequently the organic element has been overlooked. The difficulty in making a differential diagnosis is very great. We would be inclined to believe that a patient were suffering from organic changes of the cord after severe trauma if he had paralysis of the lower limbs, either plastic or flaccid, according to the level of the portion damaged, if he had a sensation of compression about the waist, if the functions of the bladder and rectum were disturbed, and if the symptoms developed within a reasonable time after the accident. It is true, that even this group of symptoms may be purely functional, but it should give rise to grave suspicion of organic changes. We call that which we do not understand, functional, and the word is most unscientific. Hysteria is the chief of the neuroses, but it is difficult to explain all the manifestations of this disease on a purely functional basis. We refer to such conditions as gangrene, trophic lesions, or even death. If we accept the interesting and captivating theory of the amoeboid movement of cells advanced by Lépine, Duval, Dercum, Pupin, Lugaro, etc., and apply it to hysteria, we are forced to believe that after a time the cell loses its power of extending its processes, and that organic changes occur within it. The many examinations made by the method of Nissl have shown us how very susceptible the cell is to external con-

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and there was no history of syphilis in any member of the family. When three months old, a number of furuncles appeared on the face and neck, and starting from one of these, an attack of erysipelas developed. There was prolonged high fever, but no convulsions. Soon after this it was noticed that her hands trembled. Her speech had always been slow. Two years ago she had a severe attack of scarlatina, and for some time after this the speech was worse. She is more or less emotional. When first seen on October 17, 1896, the pupils were equal and responsive, and there was no nystagmus. There was slight tremor of the tongue; the deep reflexes of the arm were lively, and the knee-jerks were exaggerated; ankle clonus could be elicited in the right foot. There was a marked intention tremor in both arms; there was no muscular atrophy. Her voice was harsh, and the speech slow and monotonous. Mentally she was backward.

The second case was that of a girl of eleven years, who had been seen first in September, 1895. There was no history of previous nervous or mental disease in the family. Her eyes had always been very prominent. For the past year she had lost control of the bladder very largely. She had also been weak, and unsteady on her feet. She was fairly developed; there was divergent squint and a fine nystagmus. The ophthalmoscope showed optic atrophy in both eyes. The pupils were large and responsive. There was a fine tremor of the fingers when at rest, and a marked intention tremor in both hands. The gait was that of an ataxic paraplegia of cerebellar origin. The knee-jerks were greatly exaggerated, and the ankle clonus was present on the left side. Sensation was perfect in all parts of the body. The speech was not affected, and the child appeared to be quite intelligent. Ataxic paraplegia, the speaker said, could be excluded by the eye symptoms.

The third case was that of a girl of fifteen years, first seen on March 30, 1896. Shortly after an attack of what was apparently influenza, walking became unsteady and speech difficult. Since then she had been subject to slight vertigo. Her voice was harsh, and speech required an effort. There was no nystagmus, and the pupils were responsive. The deep reflexes of the arms were very lively. The gait was that of spastic paraplegia. The knee-jerks were greatly exaggerated, and there was ankle clonus on both sides. She possessed average intelligence.

Regarding the differential diagnosis, the speaker said that the following diseases would require consideration: Infantile cerebral palsy, syphilis of the cerebro-spinal axis, hereditary cerebellar ataxia, acute disseminated myelitis, tumor of the brain and hysteria. It was often very difficult to distinguish between infantile cerebral palsy and multiple sclerosis. In the

volume. The result of the latter form of growth is to develop the "giant" type of acromegaly; but M. thinks it should be remembered that in this disease, as in any other, individual predisposition plays an important part. M. had also an opportunity of making a comparative skiagraphic study of the hand of a patient with erythromelalgia, and considers that as no change in the bony parts could be found, it might be considered as proved that this disease has no relation with acromegaly.

MITCHELL.

A CASE OF CEPHALIC ACROMEGALY, ASSOCIATED WITH SYRINGOMYELIA AND CEREBELLAR TUMOR; WITH AUTOPSY.

The author, M. Bassi, reports in the *Atti della reale Accademia Lucchese di Scienze* for March 27, 1896, a complete clinical history and the pathological findings of a case of partial acromegaly, or the incomplete form known as cephalic acromegaly associated with syringomyelia and solitary tubercle in the cerebellum. He was able with much certainty to make the diagnosis of all three affections *intra vitam*, which was confirmed by the autopsy.

The syringomyelia seemed to antedate the acromegaly and cerebellar tubercle.

KRAUSS.

CONGENITAL GIANT GROWTH OF THE EXTREMITIES. Von Torday. *Jahrbuch für Kinderheilkunde*, Band XLIII., Heft 1, 1896.

Machenhauer, *Centralbl. für innere Medizin*, No. 43, 1896.

Von Torday reports the case of a girl of 6 years, the twelfth child of healthy parents, born at term, in normal labor. The deformity existed at birth, and the limbs have retained the same proportionate size during the growth of the child.

Patient thin, pale, and delicate, subcutaneous fat much diminished by lipomatous tumors small and large, to the number of five, are distributed at different points over the surface.

The abnormality consists of enlargement and deformity of the right foot, and of the left leg and foot. The right foot shows great lengthening of the external malleolus, and enlargement of the inner part of the tarsus with the great and second toes, and deformity of the three outer toes. Great toe curled upward and away from the other toes, second toe arched upward and outward. Left leg enlarged from knee down, ankle twice as large as normal. Left foot $1\frac{1}{2}$ times as long, and $2\frac{1}{2}$ times as broad as that of an average adult. Great and little toes abducted, foot shaped like a hand, nails deformed and ingrowing. The child can walk, run, and climb. A picture of the case is given. The following are some of the numerous measurements taken:—Weight, 21 kilos; length of body, 111 cm.; circumference of right knee, 24 cm.; of left knee, 26 cm.; of middle of right leg, 19 cm.; of left, 28 cm.; of right ankle, 16 cm.; of left, 21 cm.; around right instep, 24 cm.; left, 32 cm.; length of foot on sole, right, 24 cm.; left, 31 cm. He states that it is especially the epiphyses of the long bones which undergo hypertrophy, and while the anomaly is congenital, no direct heredity can be made out.

Machenhauer's case gives the following history:—

A male child of healthy parents (born at term, labor normal, except that the cord, which was very long, was looped twice about the child's neck, and contained a knot besides), presented these anomalies. Palatal arch high and narrow. Abnormal dullness under manubrium sterni, seems to indicate an enlarged thymus. There is a general tendency to dilatation of vessels, shown in tumor on right breasts, and vascular nodule on right elbow, and in dilated vessels of skin of right hand, while the whole right leg from pelvis to foot is occupied by a huge naevus. Penis shows hypospadias, the apparent orifice of the

urethra being only a *cul de sac*, while the urine escapes from two minute orifices 1cm. further back on the under side of the organ. The fingers of both hands are unsymmetrically hypertrophied. The right leg is $1\frac{1}{2}$ inches shorter, and $1\frac{1}{2}$ inches greater in circumference than the left. Its bones are bent at an angle of 135° , opening backward, and the fold so produced is occupied by a row of small nodules—keloids. Left foot normal. On right foot, only great toe free, the rest united—Syndactylism. In a furrow near the end of the mass so formed, rudimentary nails are visible. At seven months, and at one year, the child was measured again, and the hypertrophied parts were found to have approximately preserved their proportion to that of the rest of the body.

That the hypertrophy should affect as many as three extremities is considered remarkable. M. concludes his article with a consideration of the causes of abnormal growth of the extremities, giving copious references. He lays some stress on the twisting of the cord, suggesting that it may have been knotted about the deformed leg, and that the disturbance of circulation so caused may have had something to do with the conditions found. He points out that, to whatever cause we may attribute them, general giant growth and acromegaly are apt to come on at times when there is a great change taking place in the circulation—at puberty and at the menopause. The trophic influence of the nervous system is also dwelt upon. An excellent picture of the child, and measurements of the limbs are given.

C. L. ALLEN.

PULMONARY HYPERTROPHIC OSTEO-ARTHROPATHY.

Goodlee (Brit. Med. Jour., July 11 and 18, 1896) reports several cases of this affection to which he called attention long before (1890) Marie's description and classification (1895), but adds, "It is a poor satisfaction to be able to claim that one was the first to describe a particular disease, if it is necessary to add that the case was imperfectly observed, and that its real nature was not recognized. I am afraid, however, that this is true; and it is at all events clear that most of the cases which had been reported as examples of the disease, have had actual mischief in the joints, and some have suffered from enlargements of the bones."

The first case is of interest as it ended in complete recovery. "A young man had retained for some years in his pleura a long piece of India rubber tubing which had been imperfectly secured at the time of the original opening. The swelling over the wrists and knees and ankles was very marked as was the clubbing of fingers and toes, and I do not doubt that many would have thought that there was enlargement of the bones and affection of the joints. I cannot, indeed, prove that such was not the case, but the swelling quite disappeared after the removal of the tube and the closure of the empyema."

Case 2 is probably one of arthritis complicating chronic empyema, although there seemed to be enlargement of the lower end of either femur and the upper end of either tibia.

In case 3 there was some swelling of the wrists and ankles during the existence of a chronic empyema that finally closed. Although there was some clubbing of the fingers, this case could scarcely be considered as belonging to the disease in question, but is of some interest as constituting a possible transition form.

Case 4 is somewhat similar, and the author calls it polyarticular rheumatoid arthritis. The joint trouble followed a pulmonary abscess and prolonged suppurative of tubercular glands.

Case 5 the author considered to be a "genuine case." A man of 29 was said to have had a cough since infancy. For three years the expectoration had been offensive, and six months before examination

the ankles and knees had begun to be swollen and painful. From the examination a diagnosis of pulmonary tuberculosis of the fibroid type was made, but as no tubercle bacilli could be found, it was provisionally changed to bronchiectasis. Some of the interphalangeal joints, the wrists, the knees, and ankles were enlarged, and the toes were markedly clubbed, but the author thinks an enlargement of the bones exceedingly doubtful.

Case 6, a man of 25, was also thought to be typical. Over two years before examination he had an attack of what seems to have been acute pleurisy followed by cough which persisted with the expectoration of muco-pus. The fingers had been clubbed and the knee joints swollen for two years, and examination showed a similar condition of the toes and ankles; but the author is inclined to think the bones are not enlarged, and a skiagraph of the hand showed the phalanges to be normal.

PATRICK (Chicago)

THE REFLEX ARC CONCEPT IN PSYCHOLOGY.

Psychological Review, July, 1896.

The common view of the so-called "Reflex Arc" is concisely stated by Baldwin (Princeton) in his analysis of reactive consciousness as comprising three elements corresponding to the three elements of the nervous arc. First, the receiving consciousness—the stimulus, say a loud, unexpected sound; second, the attention involuntarily drawn—the registering element; and, third, the muscular reaction following upon the sound—say flight from fancied danger. Prof. John Dewey (Chicago), however, differs from the above, maintaining that it is a circle and not an arc. The following is the gist of his argument: "A sound is an act of hearing, and muscular response is involved in this act, as well as sensory stimulus, *e. g.*, posture of head and tension of ear muscles for the reception of the sound. It is just as true to say that the sensation of sound arises from a motor response as that the running away is a response to the sound. We do not have first a sound and then activity of attention, unless sound is taken as mere nervous shock or physical event, not as conscious value. The conscious sensation of sound depends upon the motor response having already taken place; it is the motor response or attention which constitutes that which finally becomes the stimulus to another act. The final "element," the running away, is sensory-motor, and is also a co-ordination, and this sensory-motor co-ordination is not a new act supervening upon what preceded, for the sound-experience must persist as a value in the running—to keep it up—to control it. The motor reaction involved in the running occurs to change the sound—to get rid of it, and is, therefore, not merely a reaction to the sound, but *into* it. It is a circuit, not an arc, and this circuit is more truly organic than reflex, because the motor response determines the stimulus just as truly as sensory stimulus determines movement, for the movement is only for the sake of determining the stimulus—of fixing what kind of a stimulus it is—of interpreting it.

He sums up his position thus: "The circle is a co-ordination, some of whose members have come into conflict with each other. It is the temporary disintegration and need of reconstitution which occasions and which affords the genesis of the conscious distinction into sensory stimulus on one side, and motor response on the other. The stimulus is that phase of the forming co-ordination which represents the conditions which have to be met in bringing it to a successful issue: the response is that phase of one and the same forming co-ordination which gives the key to meeting these conditions, which serves as instrument in effecting the successful co-ordination. They are, therefore, strictly correlative and contemporaneous." CHRISTISON.

SENSIBILITY TO PAIN.

Psychological Review, July, 1896.

Dr. Harold Griffing experimented on 63 students at Columbia University in regard to the relative sensibility of individuals to dermal pain. His tests were made with a pressure algometer and with the induction coil. In the electrical tests the two forefingers of each hand were placed in separate cups of water, and the alternating current was sent through the body from hand to hand. The pressure test was applied to the palm of the hand and forehead in most experiments. The experiments showed that the thickness of the skin and subcutaneous tissues is an important element in determining the threshold for dermal pain. But some subjects were much more sensitive, and others less sensitive than was expected from the appearance of the hand. He found that sensitiveness not only varied with the different individuals, but it also varied in different parts of the same body, *e. g.*, those having a high pain threshold for the hand were not always correspondingly sensitive to pressure applied to the forehead and top of the head. But those who were sensitive on the hand were on the average more sensitive on the head. He also found that sensibility to electrical stimulation to be quite independent to pressure sensibility. Three subjects among the most sensitive of 27, tested as to pressure on the hand and head declared that they felt no discomfort at all when the maximum strength of current was given. One student to whom a pressure of 15 kilo. on the hand and 11 kilo. on the head gave no discomfort, considered the electrical effect unpleasant when the current was of the average strength.

CHRISTISON.

LIGHT INTENSITY AND DEPTH PERCEPTION.

From American Journal of Psychology, July, 1896.

T. R. Robinson (Toronto) has recently been experimenting on Fechner's "paradox" which consists in the fact that when one eye is partially obscured by a smoked glass or other means, the closing of that eye results in a brightening of the common visual field or of an object in the common visual field; *i. e.*, a decrease of intensity of physical stimulus results in an increase of intensity of sensation.

Robinson's experiments showed that the paradoxical phenomenon occurs only when most of the light is excluded from the second eye. If, on the other hand, comparatively little light is excluded, on the closing of that eye the whole visual field appears darker. Between these limits there is for every absolute intensity what may be called an "indifference point" or point of inefficiency, at which the light admitted to the second eye has no effect upon the brightness of the common visual field.

Aubert had previously found that at a certain degree of obscuration of the second eye occurred the greatest darkening of the common visual field, and that the admission of less light to that eye had the same effect as the admission of more light, *viz.*, a brightening of the common visual field.

CHRISTISON.

Book Reviews.

STUDIES ON THE LESIONS PRODUCED BY THE ACTION OF CERTAIN POISONS ON THE CORTICAL NERVE CELL. By Henry J. Berkley, M.D., Johns Hopkins Hospital Reports, Vol. VI., No. 1, Report in Neurology, No. 3.

In this series of papers Berkeley has studied the changes produced in the pyramidal cells of the cerebral cortex and in the Purkinje cells of the cerebellar cortex, in experimental poisoning by alcohol, serum, ricin and hydrophobia in animals, and has compared these with pathological changes found in alcohol poisoning in man. He has employed the Golgi stain which he considers of value in pathological investigations. He precedes his record of results by a careful account of the methods of staining employed, and throughout the studies compares carefully normal control sections with pathological sections. He considers the subject of artefacts and physiological variations from the normal, but concludes that there are a large number of cells in alcoholic brains that are distinctly abnormal. In the pyramidal cells there were found a distinct diminution in size, a shrinkage of a vast majority of the cortical cells particularly in the outspread of the branches, certain swellings of the dendritic processes with disappearance of the gemmules and roughening of the larger processes and to a less extent of the cell body. One out of every three or four nerve elements in the brain of the three alcoholic rabbits examined showed departures from the normal in some form. The tumefactions upon the dendritic process are irregular in size and are more numerous far away from the cell. The gemmules fall off from the process at a distance from the cell, but in extreme cases the entire dendritic process may be destitute of these gemmules. Changes in the cell body of the nature of disintegration producing defects so that the body looks as if a portion of its substance had been scooped out down to the region of the nucleus were observed. No morbid alteration could be detected in the axis cylinders of the cortex. The same alterations were found in the Purkinje cells of the cerebellum though the changes were more prominent from their intensity. The loss of the gemmules was very striking because of their greater luxuriance upon the dendritic branches of these cells.

Berkley also calls attention to the changes in the blood vessels visible in nuclear stains in acute alcohol poisoning. He says (page 34): "the changes in the muscularis of the arteries are especially interesting. Nuclei are now and then absent from areas of the median wall of the vessel, and in those that remain certain abnormalities are apparent; one-half of a nucleus being understained, presenting the appearance of a vacuole, while in the other portion the chromatin particles take the stain fairly well, and the karyoplasm also receives a portion of the dye. The nuclear membrane surrounding the entire nucleus is distinct and stained. But it is in the substance of the muscular protoplasm that the lesions are more apparent and show that the cells are undergoing a retrogressive process. They no longer have their substance clearly stained. The protoplasm, too, is considerably swollen, and its receptive qualities to the dye is no longer good. The lymphatic spaces are obliterated, the outer lamina of the vessel being pressed closely against the limiting membrane of the perivascular sheath." In the veins collections of white blood corpuscles filling the interior and penetrating the walls and filling the perivenous spaces were frequently observed, and all the leucocytes within and without the vessels showed more or less evidence of degeneration. He concludes that these

changes in the circulation are important factors in alcohol poisoning. But while he admits that there is a direct action of the alcohol upon vasoconstrictor and vasodilator nerves in the body, he agrees with other observers in failing to find any vascular nerves in the blood vessels of the brain, and concludes that the mechanism controlling the cortical arteries is different from that in other portions of the body, the muscular cells being less directly under the influence of nerve control. Hence the effects of alcohol on the brain are greater than upon other parts of the body. For muscular cells under the influence of the direct action of the poison are free from any power to urge them to contract after the immediate effect of the toxic substance is passed, and therefore remain for a long time inert; the congestion of the cerebral tissue is long continued, larger amounts of poisoned blood pass through the brain, and incidentally a greater portion of alcohol is brought to the brain than to other tissues, the deteriorated serum is transuded in increased quantities; it is carried into the lymph spaces surrounding the cerebral cells, their structures are bathed in diluted alcohol and soon undergo degeneration. The formation of the many thrombotic plugs of lymphoidal elements is very remarkable and interesting, as is also the emigration of these corpuscles through the vascular walls. This study, therefore, shows that poisoning with alcohol produces decided lesions both of the blood vessels and of the nervous elements of the cerebral cortex.

In the studies upon serum poisoning in dogs, changes of somewhat similar kind were found in the pyramidal cells, namely, an increasing number of swellings upon the dendrites, a disappearance of the gemmulæ from the branches of the dendrites, a loss of the finest branches of the dendrites, especially within the limits of the molecular layer where these dendrites come into contiguity with the terminations of the nerve fibres, and end-apparatus of all descriptions belonging to the several classes of nerve fibres terminating there. Berkley believes that it is the function of these gemmules on the dendrites to gather impulses from the terminating brushes surrounding them, and hence that the loss of these gemmules destroys the functions of the nerve cells.

The changes observed in ricin poisoning are of very much the same nature, as are also the changes in hydrophobic toxæmia. These studies are accompanied by a large number of very beautiful drawings of the pathological changes described, and by a number of microphotographs which supplement and confirm the drawings.

In Part V. the pathological alterations in the nuclei and nucleoli of the nerve cells from the effects of alcohol and ricin poisoning are studied by means of the Nissl methods.

To these studies is added an interesting sketch of the intracerebral nerve fibre terminal apparatus and of the probable modes of transmission of the nerve impulses; in which Berkley shows that it is only at the free bulbous termination of the nerve filaments that we have naked protoplasm, as everywhere else some protective sheath can be demonstrated. This proves that only at a few points can the nerve forces discharge themselves from the axons to the protoplasm of other cells, these points being at the terminal arborization of the nerve filaments. The gemmulæ upon the dendrites are the only parts of the dendritic process that also present naked protoplasm; hence Berkley concludes that it is by means of the terminal bulbous extremity of the brush and the gemmulæ of the dendrite that the nervous impulses pass from one cell to another. The report closes with an interesting record of a case of asthenic bulbar paralysis, with a bibliography of this disease.

This work should be carefully studied by everyone who is interested in the pathology of the brain. Its bearings upon the symptoms of alcoholic dementia has been already developed by the author in other articles.

M. A. STARR.

BEITRAG ZUM STUDIUM DER MEDULLA OBLONGATA, DES KLEINHIRNS, UND DES URSPRUNGS DER GEHIRNNERVEN.—By S. Ramón y Cajal. German translation by Johannes Brasler, with an introduction by E. Mendel. Leipzig: Johann Ambrosius Barth, 1896.

This recent work from the pen of Ramón y Cajal consists of nineteen separate monographs. The results are obtained from investigations on the brains of the lower animals. Some of the most important statements are the following:

The sensory root of the fifth nerve divides on entering the pons into ascending and descending branches. Many of the axis cylinders from the cells of the substantia gelatinosa cross the raphe and form the central sensory tract within the internal lemniscus, while others remain on the same side of the pons. Collaterals are sent by these fibres to the motor nuclei, especially to the facialis and the vagus.

The descending root of the fifth nerve situated near the aqueduct of Sylvius, is purely motor. The large cells of this root probably have no protoplasmic processes. The cell body receives directly the nerve impulse.

Cajal has failed in every attempt to show a connection of the pyramidal tract with the chief motor nucleus of the trigeminus.

The fibres of the anterior cerebellar peduncle arise in the dentatum, though some may have their origin in the cerebellar cortex. At the point of exit from the cerebellum many of the fibres of this peduncle give off a large descending collateral or bifurcation branch. These fibres issue from the descending cerebellar tract, or lateral descending cerebellar bundle (Cajal), and are situated within the substantia reticularis crisea, internal to the substantia gelatinosa of the trigeminus. Collaterals are given by this cerebellar bundle to the chief motor nucleus of the trigeminus, to the nucleus of the facialis, to the cells of the substantia reticularis grisea, and possibly to the nucleus ambiguus and the nucleus of the abducens.

The fibres of Meynert's bundle arise in the habenula and terminate in the interpeduncular ganglion.

The axis cylinders from the cells of the lower olive either cross the raphe—the more common course—and pass through the opposite olive, or else they form part of the anterior arciform fibres. It is probable that fibres of the inferior cerebellar peduncle arise within the lower olive, but it is also probable that the olive receives axis cylinders from the cells of Purkinje.

The sensory fibres of the vagus and of the glossopharyngeus form one common root—the fasciculus solitarius. They differ from most sensory fibres in that they do not give origin to ascending branches. The gray masses accompanying the solitary bundles unite to form the commissural nucleus (Cajal). This nucleus lies between the ependyma and the most external fibres of the gray commissure within the cervical cord. Three-fourths of the fibres of the solitary bundles, after decussating, terminate within this nucleus. The axis cylinders from the cells of the commissural nucleus form a central tract close to the sensory tract of the fifth.

Fibres were observed by Cajal to pass from the motor roots of the ninth and tenth nerves to the descending sensory root of the trigeminus.

Some of the end fibres of the column of Goll terminate in the nucleus of the opposite column.

Many of the fibres of the central sensory tract arising in the nuclei of Goll and Burdach, after decussating in the raphe, divide into ascending and descending branches.

The pyramidal tract gives origin to no collaterals during its course, and loses none of its fibres, except at the decussation; at this point it sends collaterals to the lower olive.

(He is not speaking of the spinal portion of this tract.)

The posterior longitudinal bundle contains ascending fibres of the second order, as well as some descending fibres and axis cylinders from the red nucleus. Within this bundle are found: 1, fibres from Deiters' nucleus; 2, fibres from cells in the substantia gelatinosa of the trigeminus, and 3, fibres from cells within the white reticulated substance. The first group is the most important, and from these fibres numerous collaterals are given to the motor nuclei of the eye muscles.

The fibres of the vestibular nerve divide into ascending and descending branches. Some of the ascending terminate in the nuclei of Deiters and v. Bechterew; most, however, pass into the acoustic cerebellar bundle, and may be traced to the tegmental nucleus. Possibly some fibres pass to the dentatum and the cerebellar cortex. The numerous collaterals from the descending branches form the chief termination of the vestibular nerve. They enter the descending (chief) nucleus and Deiter's nucleus. Axis cylinders from Deiter's and Bechterew's nuclei either form a bundle in front of, and external to the nucleus of the abducens—the external or direct tract—or else they constitute the chief portion of the opposite posterior longitudinal bundle, the inner crossed tract.

The fibres of the cochlearis divide in the ventral ganglion into ascending and descending branches. The former terminate in the ventral nucleus, the latter pass to the posterior part of this nucleus and into the acoustic tubercle.

Axis cylinders from the cells of the ventral nucleus pass into the corpus trapezoides.

Axis cylinders of the tuberculum acousticum, Cajal believes, enter either the corpus trapezoides or the striæ acusticæ.

The remarkable nerve terminations within the trapezoid nucleus described by Held, have been seen by Cajal. He regards them as true central nerve plates.

Cajal, with other investigators, believes that the anterior pillars of the fornix arise in the cornu ammonis.

Connection of the sensory root of the trigeminus with the facial nucleus is by means of axis cylinders from cells in the substantia gelatinosa.

The fibres within the gray reticulated substance form a sensory tract of the third order, and convey sensory impulses to the motor nuclei.

The central tract of the trigeminus, vagus, glosso-pharyngeus and vestibularis is in the posterior outer portion of the gray reticulated substance.

WM. G. SPILLER.

ANOMALIES AND CURIOSITIES OF MEDICINE. By George M. Gould, A.M., M.D., and Walter L. Pyle, A.M., M.D. With 295 illustrations, etc. Philadelphia, W. B. Saunders, 1897.

The character of this work is indicated very fully by the title, and its appearance is justified by the excellent collection of a large number of interesting facts which the authors have carefully compiled and commented upon.

Beginning with genetic anomalies, all sorts of abnormalities due to defective or abnormal development are described. Then a number of chapters are devoted to the surgical anomalies, of the thorax and abdomen, as well as of the genito-urinary system. In several chapters anomalous types of diseases of varying kinds are recorded, including such as are by this time rather well known, viz., acromegaly, myxedema and trichinosis. But in every instance some rather unusual feature of these diseases is indicated.

Neurologists will be particularly interested in the records of unusual spinal deformities, of unusual gaits in extreme cases of infantile spinal paralysis, possibly also of the unusual cases of obesity, and of hemi-hypertrophy.

Chapter XVII. on anomalous nervous and mental diseases, including such well known states as facial hemiatrophy, saltatoric spasm, *astasia-abasia*, will be of less importance to the specialist on nervous and mental diseases. Certain forms of disease may prove to be "curiosities" for the ophthalmologist, which are not even great "rarities" for the neurologist.

We have no fault, however, to find with the character of the work, and think that on the whole it was well that such a work should have been undertaken. If it will have served no other purpose than to do away with the reports of "rare" cases which are not rare, it will have fulfilled a very worthy mission.

The book is well written, carefully illustrated, well printed and contains much of interest and much that is instructive for every medical man, be he general practitioner or specialist. B. S.

BOOKS RECEIVED.

Clinical Lectures on Mental Diseases. T. S. Clouston, M.D., Edin. F.R.C.P.E., Physician-Superintendent of the Royal Edinburgh Asylum for the Insane, etc. Colored plates. Lea Brothers & Co., Philadelphia, Pa.

The Year Book of Treatment for 1897. Lea Brothers & Co., Philadelphia, Pa.

The American Year Book of Medicine and Surgery. George M. Gould, M.D. Illustrated. W. B. Saunders, Philadelphia, Pa. Cloth, \$6.50; one-half morocco, \$7.50.

Inebriety; Its Source, Prevention and Cure. By Charles Folhn Palmer. Fleming H. Revell Co., Chicago, Ill.

Swedish Movements or Medical Gymnastics. By T. J. Hartelius, M.D., and translated by A. B. Olsen, M.D., with introduction and notes by J. H. Kellogg, M.D. Modern Medicine Pub. Co., Battle Creek, Mich.

A System of Practical Medicine by American Authors. Edited by Alfred Lee Loomis, M.D., LL.D., and William Gilman Thompson, M.D. Volume I., Infectious Diseases. Illustrated. Lea Brothers & Co., Philadelphia, Pa.

State of New York. State Commission in Lunacy. Seventh Annual Report, Oct. 1, 1894, to Sep. 30, 1895.

THE
Journal
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Original Articles.

COMPRESSION PARAGLEGIA IN POTT'S DISEASE OF THE SPINE.

Based Upon an Analysis of Seventy-Four Cases.

By V. P. GIBNEY, M.D.,

ON the third of December, 1877, a little overing to this society a paper on the Paralysis nineteen years ago, I had the honor of presenting of Pott's Disease, Based Upon a Study of Fifty-eight Cases. The paper was published in the Journal of Nervous and Mental Diseases for April, 1878. In the present communication I have endeavored to tabulate cases subsequent to 1877. Since the publication of the first paper I have presented to the Section on Neurology of the New York Academy of Medicine, October 9th, 1895, a paper on Compression Myelitis of Pott's Disease Treated by Large Doses of Potassium Iodide. Within the last few years a decided impetus has been given to the interest in the subject by the publication of a number of cases in which laminectomy has been performed for the relief of the more obstinate paraplegias, but I have left the analysis of these cases to gentlemen who will follow me this evening, and shall endeavor to give you as accurately as possible the facts which I have been enabled to secure by a record of final results obtained without the assistance of operative procedures.

To one who is accustomed to statistical work and to the search for final results, the study of cases operated upon is far from satisfactory. The mere mention of a certain number of cases cured by this operation or that prompts one to look closely into the detailed history of the cases themselves. The observations are not always accurately recorded, the reasons for operation are not sufficiently explicit, and the results themselves correspond very closely with the results obtained by non-operative procedures. I make these statements, not to forestall any discussion of the subject or prejudice any one against the attempts at relief in what are known as desperate cases. All authors who have written upon this subject agree in relegating to laminectomy patients who have failed to get relief from the ordinary methods of treatment, and who seem to be hopelessly incurable. In looking over the literature, I find cases of incomplete paraplegia operated upon, cases that seemed to be already recovering; but there are a certain number where the operation was done with extraordinarily good results. The President of the Neurological Society has seemed desirous of obtaining all the information of a practical nature that is possible, and the standing of the gentlemen who are to follow is a guarantee that the work assigned to them will be well done. We ought, therefore, to know at the close of this discussion just what cases are suitable for operation, in what cases an operation is imperatively demanded, and in what cases an operation will be of no benefit whatever.

The question is often asked whether I still believe in the great value of large doses of potassium iodide, and I shall answer, not by statistics, but in a general way. In the first place, I am not wedded to any one form of treatment. I believe that the potash is an excellent adjuvant, and that it is much more efficient when perfect immobilization of the column is secured. I am also convinced that a case will recover more promptly if these two elements of treatment are combined. As a matter, therefore, of routine practice, I employ the drug in large doses up to the point of tolerance. I employ the Taylor brace in some instances, more frequently, however, the plaster-of-paris corset, with a head spring or chin rest. I sometimes resort to the wire cuirass in very young children, to the Cabot, or Bradford frame at times, and in a great many instances

I employ the Paquelin cautery. The management of an individual case depends largely upon the conditions present. If one can secure rest in bed, on a frame, with traction by the head, the patient, I am convinced, will progress more rapidly. I am still opposed to the use of electricity in any form, and can see no good reason for its employment.

In studying the cases for analysis on this occasion, the temptation to present many by way of illustration is difficult to resist. Of a fair number I have secured pathological information, which, however, is but confirmatory of the work done by many of my predecessors. The consensus of opinion seems to be against the employment of the term compression myelitis, and in favor simply of pressure paralysis. Yet there are instances where a myelitis does exist where cord changes remain permanent and where any form of treatment is necessarily of little avail. If we accept the pressure theory as the best working theory, it is easy to understand how the enlargement of the lumen of the canal by removal of the posterior arches is a commendable operation. The advances made in cerebral and spinal localization enable us to tell pretty accurately just what parts of the column are involved, just what tracts are encroached upon, and it is often possible to determine without an exploratory incision how extensively the cord is diseased.

One of the patients, a girl, twelve years of age, died April 15th, 1896, after a pretty complete paraplegia of six months' standing. She died of tubercular meningitis, and I was fortunate in securing an autopsy conducted by Dr. Henry S. Stearns, who made this report: "The cord just above the curvature shows a sclerosis of all areas of white matter, least in the posterior columns and greatest in anterior columns and direct cerebellar and Gowers' tracts. Moderate in the crossed pyramidal, antero-lateral and mixed lateral tracts. Below the curvature there is much less sclerosis generally, but the posterior columns and the descending antero-lateral tract are almost completely destitute of nerve fibres, while all of the anterior and remainder of lateral columns present only very moderate evidences of sclerosis. These central lesions are decidedly confused as to their distribution, which is partly, at least, accounted for by a considerable amount of productive in-

flammation under the dura mater for about two inches in the region of the curvature. There are also some small patches of new formation, sub-meningeal, above and below the curvature. These latter are isolated, and, of course, would give rise to some of the otherwise unexplainable areas of degeneration and sclerosis."

I am well aware that statistics, as a rule, are uninteresting; that is, for the reader of a paper, but I am sure that their value is appreciated by men who are writing books or cyclopædia articles, and hence I offer no further apology for presenting the data I have secured in statistical form. I have about ten tables, which will be represented in the customary order:

TABLE I.—SEX.

	MALE.	FEMALE.	WHOLE NO.
From recent statistics.....	33.....	41.....	74
From former paper.....	30.....	28.....	58
Total.....	63.....	69.....	132

It will be seen, therefore, that sex offers no predisposition.

In the following table I have endeavored to give the location as nearly as possible, and while the regions are mentioned, it must be remembered that the deformity frequently extends from one region to the other. The location, then, means the region in which the greatest deformity appears.

TABLE II.
Location of Bone Disease.

Cervical	5
Upper dorsal.....	28
Dorsal	41
Lumbar	0
Total.....	74

This table corresponds very closely with some observations I made in 1877 and published in the paper to which reference has already been made. I take the liberty of incorporating the following quotation: "From the notes of 295 cases of Potts' disease, I find sixty-two producing paralysis more or less complete. The number wherein the disease was situated above the mid-dorsal region was 189, and in this group the sixty-two paralytics are included, only three or four being associated with disease involving

the lumber vertebræ, about one-half of the patients that have been affected with caries in the cervical or upper dorsal regions were at one time paralyzed, while nearly one-fifth of the whole number, irrespective of locality, were thus affected."

In citing the cases for the present paper I found a few recorded as paraplegic, and I found a few cases of lumber Potts recorded as paraplegic, but on noting them closer I was able to eliminate the paraplegia altogether. Last notes as to these, "unable to stand," "unable to walk," were found, but the reason for this inability to stand or walk was discovered to be the faulty position of the limbs, by reason of psoas and iliac abscess or psoas contraction, involving one or both limbs. I have carefully tried, therefore, to incorporate those only where the paraplegia was indubitable, as shown by the exalted reflex spasm and inability to move the limbs, even when the patient was sitting or lying down. I am confident that a compression affecting the lumber cord will not produce a genuine paraplegia.

TABLE III.

The Degree of Deformity.

Much deformity.....	49
Little deformity.....	25

This gives one a general idea of the nature of the paraplegia, and helps to disabuse one's mind that the angular shape of the bosse is the prevailing factor in the production of the paralysis. A small number of my cases had no deformity whatever, and, indeed, cases are not at all rare where no deformity whatever existed, and yet the compression paraplegia existed. These have been demonstrated by autopsy.

One of the most aggravated cases that has ever come under my observation was one of this kind. It was in a boy who was admitted to the hospital the 28th of March, 1890. He had been under observation in Out-Patient Department for more than a year prior to this date, suffering from a tuberculous ostitis of the hip. The disease was under very good control. The deformity was very slight, and we had every reason to suppose that the resolution was the legitimate outcome of the treatment. A month prior to his admission he complained of a little pain in his

back, and on examination there was found to be tenderness on concussion of the upper part of the spine, recognized by striking the top of the head as he stood. There was also reflex spasm on moving the head and pain in the occipital nerves. A diagnosis was made of cervical Potts and admission was advised. A day or two after admission it was found that he had a little exaltation in the reflexes, both at knee and ankle, in the sound limb. We at once suspected incipient paraplegia and put him to bed with traction to his head. Sand bags were also applied on either side of the head, so as to give as much rest and protection to the parts as possible. He, nevertheless, developed complete paraplegia. The case progressed from bad to worse, and it became an exceedingly difficult problem how to prevent deformity at the diseased hip and also deformity in the cervico-dorsal region. Reflex spasms became intense, and for two or three years we were obliged to keep him, a greater part of the time, in plaster-of-paris from the axillæ to the balls of the feet. When he was not in plaster, traction was made on the limbs by weight and pulley, and counter-traction at the head, by the same means. It was fully two years, therefore, before any amelioration in his paraplegic symptoms was noted. During this time he suffered from incontinence of urine; occasionally, incontinence of fœces and sacral decubitus. It was next to impossible to apply plaster-of-paris without getting excoriation and finally a wire cuirass was employed, but the spasm was so great that pressure sores were induced. A recumbent posture was maintained all the while, even for one or two years longer, so that his treatment extended over a period of four years. The potassium iodide was employed in large doses. The pressure ulcers would heal under special dressings, and, finally, his paralysis began to recede. Then we had to resort to tenotomy and myotomy and various stretchings under ether or gas in order to get the limbs into good position. It was necessary to perform circumcision on account of a very severe form of ballanitis. After all, I am able to count this case as one among the improved, indeed, I might count it as one among the cured but for the difficulty he had in getting about by reason of the deformity of hip. It is interesting to note that after this prolonged siege there is no deformity in the cervical dorsal region. In other words, he has passed through

all the stages of Potts' disease, we may say, without deformity.

As further illustration of the little influence that deformity has upon the paraplegia, let me cite another case in which recovery occurred promptly without any recession of the deformity:

A girl, eight and a half years of age, was admitted to the hospital the second of May, 1883. She had suffered from Potts' disease of the spine for four years. The bossé included nearly all of the dorsal vertebræ and was very conspicuous. Shortly before her admission she became paraplegic. The treatment employed was a simple Knight spinal brace, which made no claim whatever to posterior pressure, but was a mild form of fixation. She began to improve within a few weeks after admission, and at the end of eight months was entirely restored. Letters received for a few years subsequent to her discharge confirmed the permanency of her cure.

TABLE IV.

Interval between recognition of Bone Disease and appearance of Paraplegia.

Simultaneously	14
Within 3 months.....	7
Within 6 months.....	15
Within 12 months.....	18
Within 2 months.....	9
Within 5 months.....	8
Within 7 months.....	2
Within 14 months.....	1
Total.....	74

In many instances the symptoms of bone disease are not recognized until the deformity appears. The symptoms may often exist, to a typical degree, and yet the proper interpretation is not given. Very good practitioners fail to recognize the disease until the deformity has arisen. They are so in the habit of looking for "malaria" and "rheumatism" and "neuralgia" and "growing pains," that symptoms which ought not to be overlooked, and which ought to be correctly interpreted, pass unnoticed, until the mother herself calls attention to the Potts' disease.

I take this opportunity, therefore, of making another appeal for the early diagnosis of Potts' disease of the

spine, merely suggesting that an examination of the child naked will assist materially in making such diagnosis.

TABLE V.

Completeness or Incompleteness of the Paraplegia.

Complete	65
Incomplete	9

Among the cases of complete paraplegia, there were several instances of dorsal and sacral decubitus, and eight cases where the incontinence of urine was a most annoying feature. In a few instances incontinence of fœces existed. The treatment was varied. The majority of the patients wore, at one time or another, a plaster-of-paris jacket. A certain number wore a spinal brace, sometimes a Knight brace, sometimes a Taylor posterior spinal assistant. A few were confined to bed on a frame, with traction. In a certain percentage the potassium iodide was employed in large doses. The cautery was employed in adolescence, in addition to the protection and the potassium iodide. I have not employed the cautery to any extent in children, for the reason that the thought of being burned is so repellent that I have contented myself with other means. It is unnecessary to give an elaborate display of the different methods of treatment, because I am not prepared to state definite results therefrom. I thought it best to make two general classifications; one giving cases treated by potassium iodide and an immobilization, and another class treated by some form of immobilization not always complete. In separating these two, I find that I can present a table which will give approximate results:

TABLE VI.

Immobilization only.

Cured	18	\$64.28
Improved	2	\$ 7.14
Unimproved	4	\$14.29
Died	4	\$16.29
Total.....	28	

For comparison, let me present another table:

TABLE VII.

Immobilization and Large Doses of Potassium Iodide.

Cured	27	\$58.69
Improved	10	\$21.71
Unimproved.....	4	\$ 8.70
Died	5	\$10.87
Total.....	46	

Before proceeding further with the final results, I deem it best to present a table which will give the duration of treatment and also one that will give the interval between the discharge of the patient from the hospital and the last recorded note. This may be further supplemented by a table giving the duration of the paralysis.

TABLE VIII.

Duration of Treatment.

Under 1 month.....	8
Under 3 months.....	8
Under 6 months.....	14
Under 12 months.....	20
Under 2 years.....	9
Under 3 years.....	5
Under 5 years.....	5
Under 5 years.....	2
Under 6 years.....	3

TABLE IX.

Interval between Discharge from Hospital and Last Note.

No interval.....	32
From 1 to 6 months.....	10
From 6 to 12 months.....	6
2 years.....	4
3 years.....	3
4 years.....	4
5 years.....	3
6 years.....	3
8 years.....	1
9 years.....	3
10 years.....	2
12 years.....	1
13 years.....	2

With regard to the number of cases in which there was no interval, I would like to state that a number of these were heard of afterwards, but I was unable to fix a date and was unable to find any record in the histories. It is fair to presume that a case once cured and so recorded will not relapse under a moderate degree of protective treatment. We have found that when the cases do relapse, there is some very good reason therefore, such as the premature removal of the apparatus, extra strain or impaired health.

TABLE X.

Duration of the Paralysis.

Three months and under.....	5
From 3 to 6 months, inclusive.....	12
From 6 to 9 months.....	8
From 9 months to 1 year.....	9
From 1 year to 1¼ years.....	4
From 1¼ years to 1½ years.....	6
From 1½ to 2 years.....	11
From 2 to 2½ years.....	6
From 2½ to 3 years.....	4
From 3 to 3½ years.....	1
From 3½ to 4 years.....	1
From 4 to 5 years.....	2
From 5 to 6 years.....	3
From 6 to 7 years.....	1
10 years.....	1
Total.....	74

I have not included in this list any cases that are at present under treatment, but have purposely omitted such. The case in which the paralysis existed for six years is worthy of note, as it was a most remarkable one, by reason of the extraordinary spasm, prior to and during the early part of the active treatment.

This was in a boy ten and a half years of age, who came under my observation Oct. 22d, 1891. He was referred to me by Dr. William H. Thompson, of this city. His Potts' disease dated from an attack of the grip in February, 1890. Ten months later, while wearing a plaster-of-paris jacket with head support, he manifested weakness in the upper and lower limbs. A month later he was unable to walk. The treatment employed at this time was traction and counter-traction, in the recumbent posture, and was carried out for two months most successfully. During this period the paralysis became profound. The upper extremities became paralyzed first. The reflex spasm was confined to the thigh flexors. In July, 1891, he was removed from bed and subjected to baths at the Hot Springs, Virginia, for seven weeks without any amelioration. At the time of my first observation his was a frail-looking body, chest sunken, muscles considerably wasted, bony prominence marked. He could not turn from side to side, but lay on his back and toward the side with the left thigh flexed strongly on the pelvis at an angle of 90°, the leg being flexed sharply on the thigh. The spasm was so great that the heel rested against the buttocks. The right

limb was held in moderate spasm, but he could, with difficulty, overcome the spasm himself. When he got the thigh and leg down to about 135° the whole limb would become suddenly extended to 180° , with a distinct snap like the trigger of a gun. At this time the thigh muscles were all tense; leg muscles the same; foot fully extended. The bladder sphincter was very weak, but he had control over his rectal sphincter. There was no reflex spasm in the upper extremities. The right hand grasp was very weak, but it was a little stronger in the left. The deformity of his spinal column was not very marked. Disease was located in the cervico-dorsal region. The treatment adopted was the cautery, a frame and potassium iodide, supplemented by the potassium bromide at night.

By the 13th of November there was a little relief in the spasm, but it was still very annoying at times, requiring hypodermics of morphia to secure sleep. A note made Nov. 30th states that he had reached ninety-five grains of potassium iodide three times a day. He rested better at night. The exacerbations were not so frequent and not so severe. He had very good control of the sphincter of his bladder. On Dec. 5th I gave him ether and fully extended the left limb. Weight and pulley was employed to maintain the extension. At this time he was taking one hundred grains of potassium three times a day. The improvement continued with very little interruption, and by the 21st of Dec. I gave him smaller doses of the potassium, employed as an adjuvant the hot water douche at a temperature of 112° . It was not until the 28th of March that any eruption from the iodide appeared. On the 4th of June, 1893, he was discharged from the hospital and removed to his home in a Western city. He was taken home in a wire cuirass, in which the limbs were easily held in full extension, the muscular spasm being very infrequent and very slight. I received letters from time to time, and on the 14th of November, 1892, when I saw him at his home, I found that he had gained flesh perceptibly, that the spasm was very slight, and that his pains were trifling. He slept well for three or four hours at a time, was taking no opiates or narcotics, but there was no appreciable power in the lower limbs. I saw him again a year later, when there was still no return of power. He was very comfortable and practically well but for the paraplegia. This condi-

tion continued, and on the 20th of Nov., 1896, he died from some intercurrent affection. There was no autopsy.

In estimating the results I am able to present the following table:

TABLE XI.

Final Results.

Cured	45	\$60.80
Improved	12	\$16.22
Unimproved	8	\$10.82
Died	9	\$12.16
Total.....	74		

I am thoroughly convinced that the improved cases will recover, or have already recovered, but I am unable to get at the facts in the cases. I have simply put them under the heading "improved." In tracing out the causes of death, I find that in the nine patients who died during the course of the disease the causes are shown in the following table:

TABLE XII.

Cause of Death.

Myelitis	3
Tuberculosis	4
Capillary bronchitis	1
Unknown sudden death.....	1

A further table may be added which will show what became of the nine cured cases; that is, nine cases cured of the paralysis and so recorded. These died later of the following diseases:

TABLE XIII.

Cause of death in Patients subsequent to the Cure of the Paralysis.

Unknown (two dying suddenly).....	4
Tuberculosis	2
Pneumonia	1
Alcoholism	1
Pott's disease with suppuration.....	1

Before concluding this paper I desire to place on record an extraordinary case of rotary lateral curvature of the spine, involving the upper dorsal region. I am prompted to do this because of a patient at present under observation with lateral curvature of the spine, right side, and a marked degree of ataxia. One often wonders why we do

not get paralysis in cases of high degree of curvature, why the cord itself must not necessarily be compressed by the sharpness of the curve.

The case I report is in a boy who was fifteen years of age at the time of the development of the paraplegia symptoms. He first came under my care on Oct. 21st, 1891, and was referred by Dr. G. H. Fox, of Rutland, Vt. His curvature had been observed six months prior to the above date. He had no pain or inconvenience at the time and was in very good general condition. At the time I first saw him his thorax was in moderate pigeon breast; the lateral curvature was not great, but still noticeable. There was associated with the lateral curvature an antero-posterior curvature, which was less than an inch in height. The actual lateral deviation was less than one-half an inch, but the rotation was rather sharp, while the curve was proportionately small. I thought his a good case for gymnastic exercises, and taught him a number of movements which I fancied would be of assistance in correcting the deformity. He seemed interested in the work and on Jan. 12th, 1892, I made a note that he had been quite faithful. There was, however, no improvement that I could detect. I was forced to note a little increase in the rotation.

June 3d, 1892, I noticed that he was not exercising with any degree of energy and that it was a task for him to exercise. A few days later I fitted him with a plaster-of-paris corset and provided a swing for him to use night and morning. Oct. 20th, 1892, it was noticed that he was gaining in height and that the deformity was not increasing.

March 15th, 1893, because of sluggishness in gait, I called Dr. M. Allen Starr in consultation, and he found diminution in all the skin reflexes, exaggeration of the patella tendon reflexes, slight ankle clonus, no anæsthesia, no apparent paralysis, no rigidity. With the eyes closed the boy swayed a little. The mechanical irritability of the muscles was normal. There was some atrophy of the trunk muscles, especially the pectorals, which was out of proportion to that of the arms and legs. Dr. Starr made a diagnosis at that time of compression of the upper dorsal cord, probably below the second dorsal segment, and above the seventh, as the epigastric reflex was normal and the ab-

dominal reflex was exaggerated. He approved of apparatus which would fix and support spine and suggested a hot and cold douche to the back at night. I applied a spinal brace with a chin piece for the head, and ordered a continuance of traction by means of the swing. June 10th, 1893, he presented a marked exaltation of the reflexes, was much more sluggish in his movements and seemed to take very little interest in the treatment. There was no paralysis at this time. Oct. 19th, 1893, he was carefully measured and his limbs were found equal in length, equal in size. When he closed the eyes he swayed a little from side to side, both in standing and walking. He had been taking, since the 12th of June by my orders, potassium iodide, and when he reached thirty grains three times a day he had a troublesome diarrhoea, which was controlled after a discontinuance of the potash. April 28th, 1894, his deformity was greater. The chest walls were flattened on the right side in front, and the ribs formed a sharp ridge over the projecting side. The plane of the scapula on the right side formed an angle of 110° with the verticle axis of the body. The limbs were weak, but he was walking about. I made an attempt to fit him with a steel apparatus, but failing in this I applied in May a slight plaster-of-paris jacket.

I heard from him occasionally, but did not see him until Oct. 20th, 1894, when he was decidedly worse. His gait was shambling and unsteady. He used a cane. Drs. Starr and Shaffer on my suggestion were called in consultation at the time and each examined him very carefully. Dr. Starr found increase of the reflexes below the thighs, but no cremaster reflex. There was increased loss of power in the limbs. He made out a lateral sclerosis, but was unable to assign any cause, thinking it probably idiopathic. He had reluctantly abandoned the compression, because of the excellent support which the boy had had and because of the traction to which his spine had been subjected. Dr. Schaffer ruled out Potts' disease and was unable to explain the increasing loss of power or the lateral sclerosis as due to the curvature itself. He decided that all braces be dispensed with for the present, that the boy go to bed for three months and make no attempt to walk during that time. After a little further consultation we agreed to urge this treatment, adding thereto, on Dr. Starr's sug-

gestion, the use of farradism to the back muscles and belladonna and ergot internally. The prognosis was gloomy enough. The treatment, however, was carried out most successfully under the immediate direction of Dr. Thompson, of Rutland.

Jan. 27th, 1895, I visited the boy and found him very much improved in appearance, in general health and in muscular development of the upper extremities. The spinal deformity was certainly much less marked. He had some tenderness over the spinous processes in the upper dorsal and cervical regions. The thorax in front was in excellent shape. The patellar tendon reflexes and the ankle clonus were much aggravated. In fact, he had at this time all the symptoms of compression myelitis with paraplegia. He could move the feet about in bed, but this act seemed to bring on spasm. The sensation was good and the muscles were well developed. There was no localized atrophy. It was reported to me that during the first month of his bed treatment the feet were quite cold, subjectively and objectively, but latterly they had become warm, although the circulation in the toes seemed poor. He had borne the confinement so well that I ordered him to remain in bed three months longer. On June 29, 1896, the father reported that he was walking about with a little assistance, that his limbs seemed to be growing stronger every day. Was having massage twice a week.

He came to the city on the 17th of December, 1896, and I found him walking without support for at least a month. During the two weeks prior to his visit, the improvement had been most rapid. He could go up and down stairs and had lost that sense of weakness in his back. The deformity of his spine was no worse. The muscles about the chest and back were better developed. There was a little tenderness in two or three points over the spinal column. He could move his limbs in all directions and all the muscles were in good condition. He could stand and jump on his heels without pain. There was still a little exaltation of the patellar tendon reflexes and a little foot clonus, especially on the left side. I advised a continuance of the treatment, discouraged the use of a brace of any kind and gave a good prognosis. Indeed, this was quite unnecessary, as the progress of the case itself made this a foregone conclusion.

A CONSIDERATION OF THE PARAPLEGIA OF POTT'S DISEASE WITH ESPECIAL REFER- ENCE TO THE RESULTS OBTAINED BY ME- CHANICAL TREATMENT.

An Analysis of Forty Cases Occurring in the Service of the
New York Orthopædic Dispensary and Hospital.

By NEWTON M. SHAFFER, M.D.

When your President kindly invited me a few weeks ago to participate in the discussion of the subject which brings us together to-night, and the part was assigned me of speaking upon the Results of Mechanical Treatment, I found, after making the effort, that the time was too short to make a comprehensive digest of a large number of cases. Dr. T. Halsted Meyers had, however, already made a brief and concise contribution to the subject,¹ in which he says regarding the 1,570 cases which he investigated that it was "A number far too large to tabulate." It seemed to me, under these circumstances, that a critical analysis of the available cases occurring in the practice of the New York Orthopædic Dispensary and Hospital and a somewhat rigid scrutiny of the results obtained, would be of service, and with this end in view, and with the kind and most persevering assistance of Dr. P. H. Fitzhugh, of the dispensary staff, I have been able to collect a sufficient number of cases to aid us, I think, in our deliberations.

In approaching the subject I instructed Dr. Fitzhugh:

1st. To report all the cases available occurring in the service of the institution which had been under its continuous care and which had afforded enough co-operation at home to make the treatment reasonably effective.

2d. All such cases, "good, bad or indifferent," were to be tabulated and reported in full.

¹ "The Prognosis of Pressure Paralysis." The Times and Register, Nov. 29, 1890.

3rd. Under no circumstances were the cases to be selected and none, however bad the result, were to be rejected.

The number of cases which we are able to report, in the limited time at our disposal and under these conditions, is forty. With a much longer time for preparation many other cases might be traced, a final examination made, and the result recorded. But it is fair to assume that the cases here reported represent the average results obtained by the methods pursued in the institution in its outdoor practice.

And it seems only right to say in connection with these cases that they all occurred among the poorer classes of the tenement house population; that all of them were treated at their homes under not very favorable conditions, and that they were visited at regular intervals by some member of the dispensary staff, the mother, or some more or less interested relative or friend caring for the patient between the professional visits. Under the conditions which exist in private practice the results obtained are doubtless much better.

All these patients were subjected, generally speaking, to the same treatment. That is, a Taylor spinal brace was applied, with or without the ball and socket chin piece attachment, and necessarily all were treated in the recumbent position—the patient lying on his back upon a tolerably firm mattress, without any pillow. The apparatus was adjusted to that position which would steady and support the entire spinal column without producing undue pressure at any point. After the final adjustment of the apparatus, which took perhaps several days, periodical visits were made, the pressure line of the support was inspected and the apparatus was modified if necessary. Iodide of potassium was not used and no special internal medication was employed—the main principle of treatment being to produce a practical immobilization of the spine—especially of the diseased region.

Several tables have been prepared showing the age, first symptoms, duration and kind of paralysis, location, abscess, results, etc., to which I beg to call your attention before making any extended comment on the lessons they convey.

Twelve tables in all are submitted, to which I beg to direct your attention.

TABLE No. I.

Age of Patients on admission to the Dispensary.

From two to three years.....	3 Cases
From three to four years.....	6 Cases
From four to five years.....	3 Cases
From five to six years.....	5 Cases
From six to seven years.....	4 Cases
From seven to eight years.....	5 Cases
From eight to nine years.....	5 Cases
From nine to ten years.....	1 Case
From twelve to thirteen years.....	1 Case
From thirteen to fourteen years.....	1 Case
From fifteen to sixteen years.....	1 Case
Seventeen years.....	1 Case
Eighteen years.....	1 Case
Twenty years.....	1 Case
Twenty-nine years.....	1 Case
Forty-three years.....	1 Case
Total.....	40

It will be seen that all ages are represented in this table, thirty-two being under ten years of age, three being above that age, the oldest being forty-three.

TABLE No. II.

Showing the Location of the Disease.

(With a single focus of disease.)

In the cervical vertebræ.....	2
In the cervico-dorsal vertebræ.....	3
Dorsal, upper vertebræ.....	11
Dorsal, middle vertebræ.....	14
Dorsal, lower vertebræ.....	4
Dorso-lumbar vertebræ.....	3

37

(With double foci.)

Upper dorsal and lumbar.....	1
Cervico-dorsal and dorso-lumbar.....	1
Upper dorsal and dorso-lumbar.....	1

Total..... 40

Of these forty cases thirty-three were affected in the middle dorsal, upper dorsal or cervical regions. Three had disease both in the dorsal and lumbar regions.

TABLE No. III.

Duration of disease before the symptoms of paralysis occurred.

The disease had existed

Under one year.....	in 8 Cases
Between one and two years.....	in 13 Cases
Between two and three years.....	in 4 Cases
Between three and four years.....	in 5 Cases
Between five and six years.....	in 3 Cases
Between six and seven years.....	in 1 Case
Between seven and eight years.....	in 2 Cases

Ten years.....	in 1 Case
Eleven years.....	in 1 Case
Twelve years.....	in 1 Case
Not stated.....	in 1 Case
Total.....	40

It will be noted in this table that the paralysis appeared in thirty cases after the disease had existed from a few months to five years. Thirteen occurred between one and two years, while one each occurred ten, eleven and twelve years after the disease was first discovered.

TABLE No. IV.
First Symptom of Paralysis.

More or less sudden loss of power.....	in 15 Cases
Gradually increasing weakness of lower ex- tremities.....	in 13 Cases
Exaggerated patellar tendon reflex with ankle clonus.....	in 9 Cases
"Numbness".....	in 1 Case
Not stated.....	in 2 Cases
Total.....	40

It is not always asy to determine, among the class de-
scribed in this paper, just when the first symptom of paral-
ysis occurred. A slight weakness or even an irregularity
in gait is not always noticed unless it is preceded by some
distinct traumatism. In one case occurring in private
practice I am sure the disease (seventh cervical and first
dorsal) had existed for at least two years without detec-
tion—a somewhat sudden attack of partial paralysis caus-
ing the attending physician to examine the spine. This is
especially true of the seventh cervical and first dorsal, but
much less so of the mid-dorsal and dorsal lumbar regions.

TABLE No. V.
Relation of Abscess to the Paraplegic Symptoms.

Cases with abscess.....	12
Cases without abscess.....	28
Total.....	40
Apparently benefitted by opening of abscess.....	4
Apparently not benefitted by opening of abscess.....	1
Apparently having no effect.....	7
Total.....	12
Of the seven reported as having no effect:	
Disappeared spontaneously.....	4
Appeared before the paralysis.....	2
Appeared after the paralysis, was cured.....	1
Total.....	7

This table seems to need no special comment, except that the "twenty-eight cases without abscess" might better be written "twenty-eight cases without external evidence of abscess." No doubt in some of the twenty-eight cases a small abscess existed, which either became absorbed or encysted.

TABLE No. VI.
Amount of Deformity.

Slight	14
Marked	21
Great	5
Total.....	40

Dr. Myers calls attention to the fact "that the character of the kyphosis does not affect the paraplegia." The table would seem to indicate the same conclusion. The paralysis was no greater in the cases with "great" deformity than in the "slight" cases. It was sometimes quite the reverse.

TABLE No. VII.

Showing the Variety of Paralysis, with or without Bladder or Rectal Symptoms.

Simple motor paralysis.....	24
Motor and sensory paralysis.....	16
Total.....	40

It is unusual for a sensory paralysis to develop first. There is usually a slight motor invasion, followed in some cases by a sensory disturbance later. The primary invasion of sensory symptoms, especially if accompanied by much pain, always suggests malignant disease—a benign tumor, or some traumatic invasion of the spinal canal.

TABLE No. VIII.

Duration of Motor Paralysis after Treatment was begun.
There were in all 24 cases of motor paralysis.

Of the CURED CASES the paralysis existed

Three months.....	in 1 Case
Six months.....	in 4 Cases
Eight months.....	in 4 Cases
Nine months.....	in 1 Case
Ten months.....	in 2 Cases
One year.....	in 2 Cases
Sixteen months.....	in 1 Case
Two years.....	in 4 Cases
Two years and two months.....	in 1 Case
Two years and three months.....	in 1 Case

Total.....21

Of the Cases STILL UNDER OBSERVATION, the paralysis has been present,

Two years.....	in 1 Case
Six years.....	in 1 Case
Ten years.....	in 1 Case
Total.....	3
Total.....	24

This table, which shows the length of time the patients received treatment for the paralysis, is a very important one. It will be considered in the body of the paper.

TABLE No. IX.

Duration of Sensory Paralysis (after treatment was begun) either with or without Bladder and Rectal Symptoms.

In the cured cases the paralysis existed

Two months.....	in 1 Case
Three months.....	in 2 Cases
Five months.....	in 1 Case
Eight months.....	in 1 Case
Twelve ¹ months.....	in 1 Case
Eighteen months.....	in 3 Cases
Two years.....	in 1 Case
Two ¹ years and three months.....	in 1 Case
Total	11

STILL UNDER OBSERVATION.

One ² and one-half years	in 1 Case
Three years.....	in 1 Case
Three ² and one-half years.....	in 1 Case
Six years.....	in 1 Case
Died.....	1 Case
Total	5
Total.....	16

Like Table No. VIII., this will receive attention later in a discussion of the results of treatment.

¹These two had sensory paralysis without bladder or rectal symptoms.

²These two have recovered from bladder and rectal symptoms, but have a remaining persistent motor paralysis.

TABLE No. X.

Giving the length of time in which Paralysis developed after Treatment of the Pott's Disease was commenced. The paralysis occurred, after treatment was commenced, in

One month.....	in 1 Case
Two months.....	in 5 Cases
Four months.....	in 1 Case
Six months.....	in 3 Cases
Ten months.....	in 1 Case
One year.....	in 3 Cases
One year and six months.....	in 1 Case
Two years.....	in 2 Cases
Three years.....	in 1 Case
Three years and six months.....	in 3 Cases
Four years.....	in 2 Cases
Five years.....	in 1 Case
Ten years.....	in 1 Case
Not stated.....	in 2 Cases

Total.....	27
Paralyzed when admitted to Dispensary treatment.....	13

Total.....	40
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One might infer, if mechanical treatment could cure paraplegia, that it might more easily prevent its occurrence. But this is not wholly true. In fourteen cases the patients had been under treatment one year or less when the paralysis ensued. But these patients lead a rough life—go to public school and play on the streets and are subjected to traumatism at almost every step. In one case, paraplegia developed ten years after treatment was commenced, though it must not be thought that *active* treatment and supervision was kept up all this time. Even in the best and most carefully treated cases, paraplegia may occur. Many factors bear upon this point—our uncertain knowledge of just what bony changes are going on, and whether there is a closely retained abscess near the canal, are among them.

TABLE No. XI.

DEATHS.

Of general tuberculosis (still paralyzed).....	1
Of general tuberculosis (after complete recovery).....	1
Of pneumonia (after complete recovery).....	1

Total.....	3
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Only one death occurred while the patient was still under treatment for the paraplegia. Both the others had recovered and had been free from paraplegia for some months.

TABLE No. XII.

RESULTS.

Cases treated of simple motor paralysis.....	24
Recovered	21
Still under observation.....	3
Total.....	24
Cases treated of sensory and motor paralysis.....	16
Recovered	11
Died or still under observation.....	5
Total.....	16
Total.....	40

SUMMARY.

Treated; 40; recovered, 32; under treatment, 8*.

Several conclusions may be drawn from a study of these cases and their histories as shown in the tables.

First of all comes the fact, proven by these results, that Pott's paraplegia, while a condition which demands prolonged care and attention, can ordinarily be cured in a large percentage of cases by careful mechanical treatment. And this being admitted, the question as to the detail of the mechanical support is important.

Whatever the treatment may be, it is of the utmost importance that the spine should be thoroughly "splinted." Simple recumbency with or without traction by weight and pulley from the head and feet, is not sufficient. One must bear in mind, especially in dorsal disease, that even respiration is a traumatism, and that any unnecessary movement is contra-indicated. The head traction is useless in my experience, except perhaps in high up cervical disease. The plaster-of-paris jacket, while affording some support, fails to make the uniform pressure along either side of the spine. As indicated on a previous page, the simple antero-posterior support, made of malleable steel, and which can be adjusted with absolute accuracy, forms the most rational method of protection. This can be so changed in its bearings and so modified as to its pressure that adequate support can be secured which is neither uncomfortable nor irksome. A water bed adds greatly to the effi-

*Of the 32 recoveries, 15 walk well with exaggerated patellar tendon reflexes, 5 walk well with normal knee jerk, and of the remaining 12 we have no record of the reflexes.

ciency of the treatment, but an ordinary mattress well supported by boards underneath is generally all that is needed.

We come now to a general consideration of the facts brought out by the tables.

Of the forty reported cases thirty-two recovered, three died, one while still paralyzed and under treatment, and one after recovery, both from general tuberculosis. One died from pneumonia after a full recovery from the paraplegia.

Of those remaining under observation, several, perhaps one-half, have a reasonable hope of recovery.

I might say here that in all the cases reported only one had bed sores and none had cystitis. Indeed, in children no attempt was made at catheterization and no serious inconvenience resulted. Perhaps this is the reason why there was such a uniform exemption from cystitis.

And I might say further that I have purposely avoided referring to the questions raised by the existence or absence of anæsthesia, hyperæsthesia, etc., in these remarks. The histories were too indefinite in this respect—because in some cases only gross tests were made, in others none at all.

The question may be asked, What shall be done with the four patients still under observation, who have been paralyzed from six to ten years? Shall mechanical treatment be continued indefinitely, or are they proper subjects for laminectomy?

It seems to me that in most if not all these four cases, degeneration of the spinal cord has occurred, and that an operation would be useless. It will be noticed that the longest duration of the paralysis, where recovery took place, with both the motor and sensory cases, was two years and three months, and that in all the recoveries only six got well after one year of treatment. Throwing out of the question incidental matters, such as variation in the home care, the resistance some children inevitably make to restraint, etc., it would seem that after one year has elapsed the chances of recovery diminish under mechanical treatment.

And this raises the question as to whether we are able to make an exact diagnosis in the earlier stages of paraplegia? I think we can—that is, we can judge very accurately as to the condition of the cord and its membranes, but I

think we are unable to make an equally certain diagnosis as to the seat and extent of the bone lesion, the existence or non-existence of matter under pressure. This is proven in a case which occurred in St. Luke's Hospital in 1876, under the care of Dr. Abbe. In this case an autopsy showed disease of the eighth dorsal vertebra, which had become necrotic, and a very sharp spicula of bone had been thrust forward into the canal, producing an almost complete section of the cord. Up to a certain point the man's symptoms had not been unfavorable, and the sudden onset of urgent symptoms was unexplained until after his death.

It seems to me that we are fully justified in saying that a case of simple motor paralysis should not be subjected to laminectomy. As long as the sensory tract is unaffected, surgical interference is uncalled for. And even if the sensory area is involved with vesical and rectal paralysis, the chances are very favorable for recovery without operation. If my tables are correct, the chances for recovery are at least 11 to 5 in the sensory cases, in dispensary practice, and the proportion of recoveries are much greater in private practice. In the motor paralysis the chances are 21 to 3, in dispensary practice, and the list of failures ought to be almost nil in private practice.

It is important to be able to recognize the approaching paralysis, and the condition of the reflexes is a valuable aid in this matter. Dr. T. Halsted Myers in his valuable paper⁴ says: "The first indication of paraplegia is the exaggerated knee-jerk." In nine of the cases here reported the members of the staff recognized the approaching paralysis by the existence of an exaggerated knee jerk, associated with a slight ankle clonus. It is always a safe rule to at once place a patient with Pott's disease, with these symptoms, in bed with or without a suitable support. But a simple exaggerated knee jerk—or even quite a marked condition of this kind—does not necessarily indicate an approaching paraplegia. In chronic spondylitis of any of the vertebræ above the first lumbar, the patellar reflex is often greatly exaggerated, and it may remain so almost indefinitely, with no subsequent symptoms elsewhere. But add to this even a slight clonus at the ankle and the case at once assumes a different aspect.

As a matter of illustration, I may mention the condition

⁴Op. cit.

of a patient now under observation, a child of 5 years, with disease at the second dorsal. A month ago I noticed an exaggerated patellar reflex following a simple, but somewhat prolonged cough from bronchitis. The knee jerk, already slightly exaggerated, became much more so, and a slight ankle clonus appeared. The patient was placed in bed, and strict recumbency with sand bags at either side of the body, was maintained. For two weeks no improvement occurred. I then applied a light spinal brace without a chin piece, adjusting it carefully to the outline of the spine. In one week the clonus became modified and soon wholly disappeared. If this were an exceptional case, it might not be worth recording. But I have had the same experience on several other occasions, and I venture to record it as a not unimportant clinical fact.

The point I wish to emphasize is that the prodromata of the paraplegia of Pott's disease are generally well marked, and ought to be easily recognized.

Another clinical fact might be recorded here: Whooping cough, especially if it occurs in connection with dorsal disease, is very apt to be followed by not only a rapidly increasing deformity, but it may also be followed by a persistent paraplegia. Any prolonged cough, persistent hiccough, or severe sneezing endangers the cord, especially in dorsal disease.

The relation of abscess to the paraplegic symptoms is very interesting. In a case early in my experience, involving the 7th to 10th dorsal, a somewhat sudden paraplegia was followed by the appearance of a large inter-costal abscess. Aspiration produced almost immediate relief, and a cure soon resulted. I have been interested in similar cases ever since, but the relation of abscess to the paraplegia is not a certain one. Table No. V. will illustrate this very clearly. Of the 40 cases, 12 had abscesses, and 28 had no external evidence of abscess. Four of the 12 were apparently benefitted by opening the abscess, and one was not benefitted; and in seven instances the abscess had no apparent effect on the paraplegic condition. In two cases the abscess appeared before the paraplegia, and in one case abscess appeared after recovery.

The location of the disease is very suggestive. Of the 40, 37 had a single focus of disease; 2 in the cervical region, 3 in the cervico-dorsal, 11 in the upper dorsal, 14 in the mid-dorsal, and three in the dorso-lumbar. Paral-

ysis from the lumbar disease is very rare in my own experience. Dr. Myers⁵ found 18 in 218 cases.

The remaining three had double foci of disease. One, upper dorsal and lumbar; one, cervico-dorsal and dorso-lumbar; and one, upper dorsal and dorso-lumbar. In each one of the three cases it is probable that the upper focus of disease produced the paralysis.

It would seem from Table No. III., that paraplegia may develop at any time in the history of the disease, and I have known patients who are supposed to be well to have paraplegia. In one of the cases reported upon to-night, paralysis occurred before any deformity existed. My table shows that it may occur as early as a few months after the disease has been recognized, or as late as 13 years after its first appearance. It is safe to say that no patient is free from paraplegia until the tubercular process has entirely stopped and the vertebræ are consolidated, though, of course, as the time passes, the danger of paralysis becomes less.

As regards the duration of the motor paralysis, under mechanical treatment, in those who recovered, it will be seen in Table No. VIII. that it existed in periods varying from 3 months to 2½ years in 21 cases. The average duration of the simple motor paralysis was about 7 months.

The duration of the sensory paralysis, with bladder and rectal symptoms in 9 cases, varied from 2 months to 2 years. The average duration of the paralysis was 10 months.

In two cases, with sensory paralysis, without vesical or rectal symptoms, the duration of the paralysis was 19½ months.

Of those who still remain under observation—8 cases in all—two have recovered from severe and prolonged bladder and rectal symptoms, but have remaining a persistent motor paralysis.

Of these eight cases, four represent a series of cases to which full reference has been made, the question being: Are they fit subjects for laminectomy?

In closing my remarks, I would like an opportunity to record before the Society a fact which I think has not been referred to before.

Pott's disease, in the dorso-lumbar region, as well as in confirmed rotary lateral curvature in the same region, may seriously interfere with, or may wholly arrest, men-

⁵Op. cit.

stration. I have the records of four cases which bear upon this point. The first one is a woman now about 48 years old. I first saw her over 20 years ago. Up to 20 years of age she had been apparently a healthy woman, menstruating regularly. At that age she developed Pott's disease involving the last three dorsal and the first and second lumbar vertebræ. The disease progressed rapidly, with the formation of an intra-pelvic abscess on the left side, which gave rise to no trouble and was gradually absorbed. At 25 she had ceased to menstruate, although she had maintained good general health through out the entire time, and was to all external appearances a well-developed woman. She has not menstruated since.

The second case, a woman of 25, when I saw her first about 15 years ago, had Pott's disease involving the 9th and 10th dorsal and the first lumbar vertebræ. The disease developed before adolescence. She never menstruated, although the mammary development was not below the average, and she looked like a well-developed and well-nourished woman.

The third case, involving the 12th dorsal and the 1st and 2d lumbar vertebræ, aged 35, in all external respects a fully developed woman, menstruated at intervals and very irregularly for a few years (the disease developed at 15), and after 25 or thereabouts, she menstruated only once in two or three years. And this week I examined a woman aged 43, with very marked rotary lateral curvature in the dorso-lumbar region, the curvature having existed since childhood, who has menstruated only 6 or 7 times in her whole life. In other respects, and aside from her deformity, she has been a healthy, active woman.

In several other cases I have found the menstrual function extremely irregular in women who have had Pott's disease in the dorso-lumbar region early in life.

I have never found a similar condition in Pott's disease above the 10th dorsal, though I have carefully questioned many patients whom I have examined.

These facts I have publicly mentioned on several occasions; before the Orthopædic Section of the N. Y. Academy of Medicine over five years ago, and in my lectures at the N. Y. Orthopædic Dispensary and Hospital during the past twelve years. I deem them worthy of a more permanent record and take pleasure in bringing them to your notice.

LAMINECTOMY IN SPINAL CARIES PARAPLEGIA.

By DEFOREST WILLARD, M.D.,

Clinical Professor Orthopaedic Surgery University, of Pennsylvania; Surgeon Presbyterian Hospital, Philadelphia.

It is doubtless expected by your Society that I should discuss this question chiefly from the surgical aspect, but you may discover in this as in other conditions that the surgeon is sometimes less radical in his views than the medical man.

I have had considerable experience in the treatment of these cases upon the lines of treatment by rest, by fixation of the spine, by extension in the horizontal position and by alteratives as well as by laminectomy, and am compelled to say that thorough enforcement and proper application of the methods named have been successful in a very large majority of cases in securing improvement in a year's time, and that this improvement ordinarily progresses until locomotion of good degree is finally secured.

In looking back over the work of thirty years I can recall very few cases which have remained permanently paralyzed.

The prognosis, judging from my own experience and from that of others, is eminently favorable, but improvement is to be accomplished only by the utmost patience on the part of the individual and his friends, and by untiring watchfulness and much skill on the part of the surgeon.

As I have indicated in this outline, radical surgical interference in the shape of removal of the laminæ to relieve the element of compression should not be undertaken until less dangerous forms of treatment have been most assiduously and patiently tried for a long period of time. Formerly it was my rule not to interfere until at least a year of rigid treatment had been tried; now, I am inclined to lengthen this period somewhat, and in certain cases to

continue it for at least a year and a half, even in the face of non-improvement.

I am led to these conclusions, first, by the fact that certain cases do improve even after a year's apparently hopeless treatment. Secondly, by the high mortality of the operation, as shown by statistics. Thirdly, by the fact that the temporary improvement accomplished by the removal of the tubercular mass surrounding the cord is, unfortunately, not assuredly permanent, but that an increase in this deposit may reproduce the symptoms in as aggravated a form as before the operation.

I do not condemn the operation; on the contrary, I occasionally practice it; in selected cases it has its legitimate place in surgery when other measures fail.

When the operation was first introduced, or I may say, revived, some fifteen years ago, the primary brilliant results secured by Macewen, Horsley and others, aroused the hopes of surgeons to a marked degree. My own first operations were so satisfactory as to results that I was greatly encouraged in regard to these cases which are necessarily most distressing and disheartening. The occurrence of fatal results, however, and the relapse of cases operated upon, led me to examine the statistics more thoroughly in relation to the operation itself and its final results. I must confess that I was dumbfounded when, in a collection of 134 cases, secured for me by Dr. Rhein, I found that other operators were no more fortunate than myself, and that the immediate mortality from shock was 24 per cent., and that of those who died within the first month (that is those whose lives were undoubtedly shortened by the operation) the mortality was 36 per cent.—more than one-third of all the cases.

We may, I think, also reasonably infer that the lives of those who died within the year after operation were probably shortened, and, according to these statistics, nearly one-half (46 per cent.) of the number of cases operated upon have had their lives abridged by surgical interference—a most discouraging mortality for a condition which, while serious, does not immediately threaten life.

It is true, that a certain percentage of cases will naturally die from tubercular meningitis, general tubercular infection, septic infection of the cord or system, etc., yet the number of such complications is limited.

Let us examine statistics again in regard to the bene-

fits derived from operation. It is difficult to draw absolute conclusions. A number of the cases have been placed upon record a few months after operation and the subsequent history is unknown. It is impossible to say whether the reported improvement has continued, whether relapse has taken place or whether death has resulted later.

Taking the published reports, however, which probably represent the best results, about 65 per cent. may reasonably be placed in the category of deaths or "not materially improved."

It should be remembered, however, that the condition, on the other hand, is one of great gravity, and that the operation when performed is only employed for the benefit of the most stubborn and intractable cases, whose improvement under ordinary treatment is hopeless.

The merits of the operation itself should not then be judged by the figures given in statistics. I believe in the operation for certain cases; in fact, I have performed it within the last two months, although, I regret to say, with a fatal result. The case was, however, one of stubborn severity and, in my opinion, demanded surgical interference.

When it is considered, therefore, that only the most hopeless cases are subjected to operation, we must anticipate the improving of only a small percentage of cases.

In regard to the operation itself, I presume that I have performed it as frequently for this condition and for traumatism of the spine as any American surgeon, but abroad the operation has found, especially with Kraske and Lane, greater favor than it has on this side of the Atlantic.

I have not found the operation as simple as some writers would indicate. In my first attempts upon dogs and in my later experiments with these animals in spinal surgery, as well as in operating on the human being, I have found the shock very considerable, and if the hemorrhage is great, as is often the case, shock is increased.

A surgeon who has performed the operation only in the upper dorsal region realizes but little the difficulties that are encountered in the lower dorsal or lumbar region. The great mass of muscles of the erector spinæ group bleed most profusely, and the intraspinal veins also pour out blood in large quantity, adding seriously to the already exhausted condition of these cases.

The large amount of hemorrhage is one of the objec-

tions to the raising of the osteo-plastic flap, as proposed by Urban. I have tried this procedure twice, but have encountered the difficulties already mentioned. The hemorrhage also delays the operation, which is another element in the production of shock. I do not think that the replacement of the arches compensates for the objections named.

This hemorrhage can be avoided to a certain extent by keeping close to the spinous processes in clearing away the muscles, and is still further lessened by a single incision and the cutting off the several spinous processes "en bloc," as proposed by Abbe, the connected processes being then slipped to one side to permit the laminal slope to be attacked.

I usually make the skin incision a little to one side of the spinous process to avoid cicatricial pressure afterwards. As the hemorrhage is largely venous, packing and catch forceps are usually sufficient to close the vessels and ligatures are seldom required.

The only real difficulty in the operation is the removal of the first lamina. Sharp, double-jawed, ronguer forceps work best, but Hey's, or other form of short saw may be employed.

When once the cord is exposed the other laminæ are easily cut away with narrow ronguer forceps with flat lower blade.

Removal of tubercular material about the cord should be carefully accomplished, but handling of the cord should be avoided. Each manipulation of this structure adds to the shock of the operation, and I have seen most marked alteration of the pulse and respiration during the handling of the cord. No finger but that of the operator should be allowed to touch it, and sponging should be accomplished with caution.

When there is anterior bone pressure the cord may be rolled to one side and any sharp projection removed, but extensive attack upon the body of the vertebræ must be done from the side, not across the cord space.

Frequently it is impossible to remove all of the tubercular cells, hence a relapse can only be avoided by the removal of posterior pressure and the subsequent improvement of the general condition. Opening of the dura tends to the admission of tubercular cells within the cord.

Conclusions.—

1. Prognosis in pressure paraplegia is hopeful unless the cord has been actually destroyed.
2. Laminectomy for spinal caries paraplegia should never be undertaken until at least one year of persistent treatment by rest, fixation and extension (together with alteratives, etc.), has been most patiently tested.
3. The dangers from the operation are shown by statistics to be great, 24 per cent. dying from the immediate shock, 36 per cent. within one month and 46 per cent. within one year. At least 65 per cent. either die or are not improved by operation.
4. The dangers are hemorrhage, prolongation of the operation and manipulation of the cord.
5. In spite of these risks the operation has its place in surgery and is justified in selected cases when persistent and carefully applied measures have failed.

SUBCONSCIOUS REASONING. From Proceedings of the Society for Psychical Research, Vol. II., June, 1896. By Wm. Romane Newbold.

Prof. Newbold relates the case of W. A. Lambertson, professor of Greek in the University of Pennsylvania, who in the fall of '69 was instructor in Latin and Greek at Lehigh University when he took to using his spare time in the study of descriptive geometry, and algebraic, and analytic mathematics. In the spring of 1870 he struggled with a problem for two weeks, when he finally realized that he was "bagged," and so dismissed it completely from his mind, believing that after a rest the solution would become clear. The problem was: "Given an ellipse to find the locus of the foot of the perpendicular, let fall from either focus upon a tangent to this ellipse at any point." He tried to solve it analytically and had not thought of a geometrical solution. About a week after he had dismissed the problem from his mind, he woke one morning with his eyes fixed on a geometric solution of the problem in a complete figure apparently projected on an old blackboard on the wall facing him, the board being really white. He immediately got out of bed and drew the figure on a piece of paper, and in a few minutes secured the analytic solution. This is the only hallucination he ever experienced, and he is said to enjoy a most robust mental and physical health. Newbold relates two other cases in detail of a somewhat similar nature.

CHRISTISON.

SCLEROSIS OF THE CORNU AMMONIS IN EPILEPSY.

By W. L. WORCESTER, M.D.,

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EPILEPSY is classed in text-books among functional nervous disorders. It is well known, however, that organic disease of the brain may give rise to convulsions bearing all the characteristics of epilepsy; and that sometimes such convulsions may be the only symptom of the disease.

In my service in the Arkansas State Lunatic Asylum and the Danvers Lunatic Hospital, I have examined the brains of forty-three epileptics. In only nineteen of these have I failed to find gross cerebral lesions. It is of one of these, sclerosis of the cornu ammonis or hippocampus major, that I wish to speak of on this occasion.

This condition was present, on one or both sides, in twenty of my cases. In eleven, no other abnormality was found; in nine, it was accompanied by other and more extensive lesions, which, in my judgment, had a common origin with it, and this association seemed to me to throw light on the nature of the connection between it and convulsions.

A number of observations of this kind may be found scattered through earlier medical literature, but the lesion was, I believe, first brought into prominence by Meynert, who published,¹ in 1868, brief reports of twenty such

¹*Vierteljahrsschrift f. Psychiatrie, Bd. I., S. 395.*

cases dissected by him. In regard to its connection with the symptoms, he expresses himself as follows:

"For the present, I will only remark that it is very unlikely that the primary cause of so common a disease is to be found in the equally common primary affection of so limited a region of the cortex as the cornu ammonis, while frequently enough, in addition, extensive, apparently far more essential and not so strictly localized, brain diseases are present in epileptics.

"I hold the supposition, therefore, to be more warranted, that, through certain anatomical relations, the cornu ammonis becomes involved in disease in consequence of epilepsy produced by quite remote lesions."

Since Meynert's report was published, observations of this kind have been numerous, and I am not aware that any observer who has sought systematically for this lesion in epileptics has reported a failure to find it in a considerable proportion of cases. Sommer² publishes a table of ninety observations, only one of which is personal. In this case, the patient, a man aged twenty-five at death, had suffered for ten years from attacks of petit mal. Mentally, his condition was characterized by dementia with attacks of excitement. At the autopsy, the points of both temporal lobes were found softened, and the right cornu ammonis was of cartilaginous hardness. Microscopically, the author found atrophy of nervous elements, proliferation of connective tissue, obliteration and stenosis of blood-vessels, and a considerable reduction in number of the large cells of the fascia dentata. Adopting Ferrier's view, that the cornu ammonis is the centre of common sensibility for the opposite side of the body, he believes that disease of that portion of the brain excites paræsthesiæ, which, in predisposed persons, may act in the same way as peripheral irritations in producing convulsive attacks. He finds disturbances of sensation mentioned in thirty-eight of the cases in his table, and only explicitly denied in three.

Coulbault³ has collected ninety-three cases, largely identical with Sommer's, five of which seem to have been previously unpublished. He gives the microscopical findings, by Bouchard, in one. The patient was a woman, aged 66, said to have been subject to "weaknesses" for twenty years; to epileptic attacks for six years. Daily attacks of petit mal; severe convulsions about once a month. Death from cholera. The right fascia dentata was of very hard consistency, and presented, microscopically, multiplication of nuclei in capillaries and arterial adventitia, numerous nuclei and corpora amylacea in the cerebral substance. The pyramidal cells were few in number and pigmented. Left fascia dentata healthy.

²Arch. f. Psych., Bd. X., S. 631.

³Des Lésions de la Corne d'Ammon dans l'Epilepsie.
Thèse de Paris, 1881

He formulates his conclusions as follows:

1. Lesions of one or both cornua ammonis occur in certain epileptics.

2. These lesions vary in character. The most common is sclerosis, with or without atrophy. Less frequently they consist in softening or in tumors of various nature.

3. Lesions of the cornu ammonis are frequent in epilepsy.

4. Observations show that these lesions are accompanied by various disturbances of general and special sensibility.

5. Lesions of the cornu ammonis are a cause, not a consequence, of epilepsy.

6. The histological character of the lesion seems to consist principally in hyperplasia of connective tissue, resulting in atrophy and disappearance of pyramidal cells.

Hemkes⁴ found six examples of this lesion in autopsies on thirty-four epileptics. On the ground of results of experimental extirpation and irritation of the cornu ammonis, by himself and Nothnagel, he concludes that the sclerosis cannot be the cause of the convulsions, which he is inclined to attribute to a localized encephalitis, involving this convolution among other parts. He adduces the following facts in favor of this view: In all his cases of this kind the convulsions began at an early age. In five, there was a history of preceding disease—infantile convulsions in two, typhus, meningitis and scarlatina in one each. In all, the brain weights were much below the average, either for healthy persons or epileptics.

Féré⁵ has met with this lesion twelve times. He thinks the known facts of cerebral localization hardly favorable to the view that it is the cause of the convulsions, although the association is too frequent to be considered a mere coincidence. He suggests that it may be a predominant localization of a more extensive sclerosis.

Chaslin⁶ gives an account of the histological examination of the brains of four epileptics. One presented no lesion to the naked eye; in the others there was sclerosis of various convolutions, including the cornu ammonis, which latter, however, do not seem to have been exam-

⁴Allgem. Zeitschr. f. Psych., Bd. XXIV., S. 678.

⁵Les Épilepsies et les Épileptiques, Paris, 1890, p. 441.

⁶Arch. de Méd. Expér., May 1, 1891.

ined microscopically. He found atrophy of the nervous elements, with overgrowth of neuroglia and development of spider cells. These changes existed in a minor degree in the brain, which appeared normal to the naked eye. The principal point which he makes is that the hypertrophied elements are not connective tissue, in the ordinary sense of the term, but are derived, like the nerve cells, from the ectoderm. He expresses no opinion as to the nature of the connection between the lesions and the symptoms.

Fischer⁷ found, in a man, epileptic from his seventh year, who died at the age of fifty, atrophy, moderate in degree, of the frontal and occipital lobes of both hemispheres, and of the left temporal lobe, with extreme atrophy of the left cornu ammonis. Prof. Hoffman, who made the histological examination, reports atrophy, in the latter convolution, of both white and gray substance; the different layers of the latter were equally affected. The medullated fibres entering the hilum of the fascia dentata were very few in comparison with the opposite side. The blood-vessels were healthy, and there was no appearance of a primary gliosis. He considers the lesion of the hippocampus a part of the general atrophy of the hemispheres, and is disposed to accept Wundt's explanation of the preponderance of this process in the cornu ammonis, according to which it is due to dilatation of the lateral ventricle, arising from disturbance of the circulation. At the same time, he admits that there was no conspicuous difference in the size of the ventricles in his case. He also reports, without histological examination, a case in which there was great asymmetry of the hemispheres, the right weighing 560, the left 685 grammes, with atrophy of the right cornu ammonis, and another in which, after six years' duration of epilepsy, with advanced dementia, the left cornu ammonis was found broader, flatter and softer than the right.

Bourneville and d'Olier⁸ describe a case of induration of the end of the left cornu ammonis in a young woman who suffered from convulsions in infancy, ceasing at five years of age, and returning, this time permanently, at fifteen. They give no description of histological appear-

⁷Festschrift zur Feier des fünfzigjährigen Jubiläums der Anstalt Illenau. Heidelberg, 1892. S. 127.

⁸Arch. de Neurologie, I., p. 213.

ances, and express no opinion as to the relations of the lesion to the convulsions.

Pfleger,⁹ in autopsies on forty-three epileptics, found atrophy and sclerosis of the cornu ammonis twenty-three times, atrophy without sclerosis twice. Of these cases seven were men and eighteen women. The right cornu was affected in sixteen cases (three men, thirteen women); the left in six (two men, four women), and both in three (two men, one woman). During his studies and his service as demonstrator of anatomy at the Vienna School, he had never found this lesion. Among about 300 autopsies in the poor-house (Versorgungsanstalt) at Ybbs, where these observations were made, he found it in two other cases—one, a male, of paretic dementia (bilateral atrophy and sclerosis), and a woman, aged 78, hysterical and subject to convulsions without demonstrable loss of consciousness.

He is disposed to attribute the lesion to a disturbance in the circulation of the convolution during and after the attack.

Nerander¹⁰ gives an account, which I have seen only in abstract, of five cases of changes in the cornu ammonis in epileptics. Sclerosis seems to have been pronounced in only one of them. He found similar lesions in other parts of the brain of one, and in several, degenerative changes in the blood-vessels. He is disposed to think that sclerosis and atrophy in this region may be defects of development originating in foetal life or early infancy. He thinks they have no special relation to epilepsy, as they are not infrequent in persons who are not epileptic. He found more or less pronounced changes in one or both cornua ammonis in eight out of fourteen epileptics in the Lund Hospital for Insane, and during the same time no less than twelve cases of alterations of the cornu ammonis in non-epileptics; has also found differences in size between the two cornua in non-epileptics.

The latest article of importance on the pathology of epilepsy which has come under my notice is that of Blocq and Marinesco.¹¹ These authors examined the medulla oblongata and the Rolandic convolutions in nine cases of

⁹Allgem. Zeitschrift f. Psychiatrie, XXXVI., S. 359.

¹⁰Studien öfuer Förändringarna i Ammonshornen och Närligganda delar vid Epilepsi, Lund, 1894.

¹¹Semaine Médicale, Nov. 12, 1892, p. 445.

"essential" epilepsy dying from that disease. In the cerebrum they found in every case, in specimens stained according to Marchi's method, infiltration of the perivascular sheaths with granule cells, and granules, staining black, in the cells of the superficial layer of neuroglia. These changes they believe to be the result of phagocytosis of products of degeneration. By other methods of staining they failed, in four cases, to find anything pathological; in four, there was diffuse proliferation of neuroglia, of greater or less extent, with evidence of vascular disturbance, and in one, disseminated foci of sclerosis, with dilatation of blood-vessels and punctiform hemorrhages, both in the cerebral cortex and the medulla oblongata. With the exception of this last-mentioned case, the medulla was found healthy in all. They do not allude, specifically, to sclerosis of the cornu ammonis, but conclude that the lesions described by Chaslin, in the article already cited, are not, as the latter, with Féré and Marie, supposes, primary, but are the results of the attacks, as they believe to be the case with the changes which they themselves describe. They conclude that there is, in so-called essential epilepsy, an abnormal excitability of the motor centres of the cortex, of which the anatomical basis is as yet unknown; that the exciting cause of epileptic convulsions, in some cases at least, is an auto-intoxication, and that the convulsions may have anatomical changes for their result.

It appears from the foregoing, that the preponderance of authority is in favor of the view that the lesion in question is a result, rather than a cause, of the convulsions with which it is associated.

Before proceeding to the account of the individual cases of my series, it may be well to give a general account of the lesions common to them all, and which were found surprisingly uniform.

Macroscopically, in every case observed by me, the gyrus hippocampi (subiculum cornu ammonis), as well as the hippocampus itself, was reduced in size in all its dimensions—in length as well as thickness. The subiculum did not present any very marked change of consistency, but the cornu itself was always firmer than natural—sometimes of a consistency resembling cartilage.

In carmine-stained sections, no marked histological change appears in the gyrus hippocampi, either in the cells, fibres or neuroglia, until the deeper layers pass into

the compact layer of large pyramidal cells known as the stratum pyramidale (stpy). In this layer there is, in every case, absence of most of the characteristic cells and great diminution of the area of the layer. In some cases, the cells are comparatively well preserved for a limited extent. In this case, it is always at the same point—just before the entrance of the stratum pyramidale into the nucleus of the fascia dentata (stpy, Fig. II).

The stratum pyramidale is continuous with the nucleus of the fascia dentata (nfd), and the latter contains, nor-



FIG. I.

mally, great numbers of irregularly arranged, large, multipolar cells, similar, in general appearance, to those of the former layer. In all the cases of sclerosis, the nucleus is greatly diminished in size, and contained very few of the characteristic cells (nfd, Fig. II).

The stratum pyramidale and nucleus fasciæ dentatæ in the diseased convolutions are occupied by a dense, finely fibrillated neuroglia, staining more deeply with carmine than the normal tissue. It contains numerous nuclei, but, in most of the cases, comparatively few "spider cells."

Surrounding the nucleus of the fascia dentata, except

at the hilum, is the stratum granulosum (stgr), a thick layer of small pyramidal or bipolar cells, with apices directed outward. In most cases this was decidedly thinner than in normal specimens, and in some the layer was absent in a considerable part of its extent.

The changes in the white substance can be best studied in specimens stained after Weigert's or Pal's method. In such specimens the principal changes are found in the alveus (alv). This consists, as is well known, of two layers. The more superficial, thicker and denser, is composed of fibres mostly running lengthwise of the convolu-

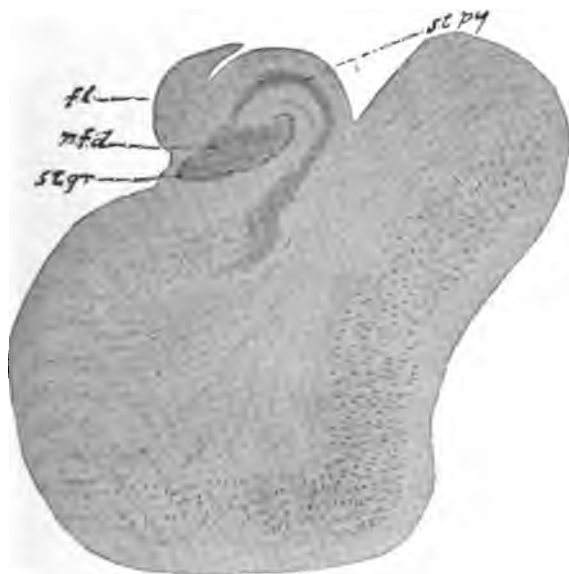


FIG. II.

tion, and, consequently, cut transversely in a cross section. This layer appears normal in structure, although somewhat thinner than in health, in the sclerosed convolutions. The deeper layer, composed of fibres cut longitudinally in cross section of the convolution, is, on the contrary, almost entirely wanting (see Figs. III. and IV., a). This layer is continuous at the hilum of the fascia dentata (hfd) with a large pencil of medullated fibres emerging from the nucleus. These fibres are also very few in number in the nucleus fasciæ dentatæ of the diseased convolutions (hfd, Fig. IV.).

The deeper layer of the alveus is contiguous to the bases of the large pyramidal cells of the stratum pyramidale. The simultaneous absence of these cells and the nerve-fibres of this layer justifies, it seems to me, the assumption that the latter are the axis-cylinder processes of the former, and that the large bundle of medullated fibres found normally in the hilum fasciæ dentatæ bears the same relation to the nerve-cells of the nucleus. It may, then, be said that the most conspicuous histological change in these cases is the disappearance of the large nerve-cells of the stratum pyramidale and nucleus fasciæ dentatæ, with



FIG. III.

the nerve fibres formed by their axis-cylinder processes.

Proceeding to the report of my own cases, I will first describe the cases in which the disease of the cornu ammonis was associated with other gross lesions.

Case 1.—E. H., white, male, aged 19 at death, an inmate of the Arkansas asylum for seven years. This was a case of infantile hemiplegia with contractures, affecting the left extremities, which were somewhat smaller than the right and much limited in movement. There were athetoid movements of the fingers and toes. He had a depressed cicatrix of the scalp, a little to the right of the

median line, deepest just anterior to the coronal suture, and extending backward for a short distance over the adjacent portion of the parietal bone, which he attributed to a blow from a falling rail when he was two years old. He was considerably demented and had frequent attacks of *haut mal*. Death in status epilepticus, July 11, 1891.

At the autopsy, on removal of the scalp, an opening, about half an inch in diameter, which had escaped observation during life, was found in the frontal bone, under the most depressed part of the cicatrix. It was circular in shape and bevelled at the expense of the outer table.

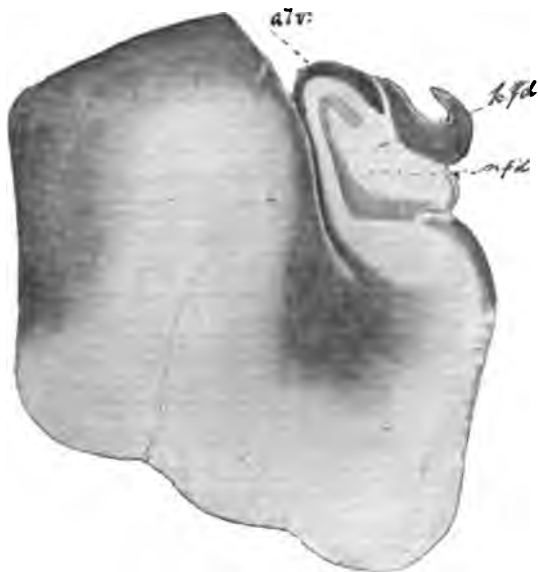


FIG. IV.

There was no depression of surrounding bone, no appearance of splintering, and no abnormal adhesion of the dura mater either to the bone or the arachnoid. The cerebral hemispheres were healthy in appearance, with the exception of the right cornu ammonis, which was much smaller and harder than the left. The right corpus striatum, optic thalamus and cerebral peduncle were atrophied, sclerotic and of a yellowish color.

Microscopically, the part of the cerebrum lying beneath the opening in the skull presented no distinct evidence of disease. The left cornu ammonis seemed healthy, but in the

right the pyramidal cells of the stratum pyramidale are wanting in its whole extent, except near the point of entrance into the nucleus, where a few are to be found. The nucleus itself is almost destitute of cells, and very much shrunk; the stratum granulosum much thinned and wanting in places. Nuclei are numerous in the neuroglia of the sclerosed portions, but spider cells wanting.

Case 2.—S. B., white, single woman, aged 46 at death, inmate of the Arkansas asylum for two and one-half years. Had talipes varus of right foot, and paralysis with contraction of both extremities of that side. She was almost completely idiotic; her vocabulary probably did not contain over twenty words, very imperfectly pronounced. Convulsions severe, but not very frequent. Death December 4, 1891, of pulmonary tuberculosis, with pyopneumothorax.

At the autopsy, extreme asymmetry of the two sides of the brain was found. The total weight of the brain, with the membranes, but without cerebro-spinal fluid, was 33 ounces. The right hemisphere, separated by Meynert's method, weighed fifteen ounces; the left, nine. The convolutions of both hemispheres were pretty regular in type, and there was no marked difference in consistency, except that the left cornu ammonis was harder in texture as well as smaller than the right. All the convolutions of the left hemisphere were small, with wide sulci. The basal ganglia of the left side were smaller than those of the right, about in proportion to the corresponding hemispheres, and the optic thalamus was of firmer consistency. Cerebellum, pons, medulla oblongata and spinal cord presented no very obvious anomaly to the naked eye.

Microscopically, the convolutions of the right side, inclusive of the cornu ammonis, show little variation from the normal condition, although there is a spot in the gyrus hippocampi which seems deficient in large pyramidal cells. In the left, the stratum pyramidale is interrupted in several places, and the deficiency in large pyramidal cells extends over the convexity of the gyrus hippocampi; the nucleus fasciæ dentatæ is almost destitute, and the stratum granulosum very deficient in their respective cellular elements. The character of the sclerosed tissue is much the same as in the previous case. In the remaining convolutions of the left hemisphere, sections from different parts fail to show any very conspicuous change in the structure of the gray

matter of the cortex, which seems to be nearly or quite as thick as in the right hemisphere. The deficiency seemed to be mainly in the white matter, in which the nuclei are more abundant than in normal brain.

Case 3.—L. M., white, single woman, aged 31 at death; a resident of the Arkansas asylum for nine years. Another case of infantile hemiplegia, affecting the left side. Upper extremity contracted, ill-developed and almost useless; lower much smaller than right and very much limited in movement. She was subject to not very infrequent attacks of grand mal and to occasional seizures in which, without loss of consciousness, the extremities of the affected side would be, for hours, agitated by clonic spasms. These could be checked by a full dose of chloral hydrate. No history of the origin of her paralysis was furnished. Mentally, she was irritable and imbecile, but had no defect of speech, and was able to do a little work. Without being known to have had recent convulsions, she passed gradually into a comatose state and died January 10, 1892.

The autopsy revealed a very similar condition to that found in the previous case. The brain weighed 37 ounces; right hemisphere, separated by Meynert's method, nine ounces; left, seventeen ounces. The right hemisphere was everywhere of much firmer consistency than the left; the temporal lobe, especially, of almost cartilaginous hardness. The basal ganglia were smaller on the right side than on the left, in about the same proportion as the hemispheres.

Microscopically, in the right hippocampus, there is almost complete absence of nerve-cells in the fascia dentata, both in the nucleus and the stratum granulosum. The stratum pyramidale is interrupted at one point, near its origin, and is ill-developed throughout. In the hemisphere in general, the cortex does not seem much, if at all, thinned, but there is apparently some deficiency of nerve-cells, more especially in the fifth layer. Spider cells are abundant, particularly in the white matter. Nerve fibres are abundant in the cortex, as shown by Pal's staining, but the tangential fibres of the superficial layer are much finer than usual.

Case 4.—T. M., white, male, aged 14 at admission, resident of Arkansas asylum seven years. Case of infantile hemiplegia, with epilepsy; duration and origin not stated. Right extremities slightly smaller than left; arm and hand contracted; athetoid movements of fingers; spastic

gait. Had an attack of dysentery, from which he seemed to recover, but failed in strength and died April 15, 1894.

At the autopsy the cranium was found to be asymmetrical, the left side being distinctly smaller than the right. The left hemisphere of cerebrum was smaller in all dimensions than the right; temporal lobe and insula distinctly firmer in consistency; cornu ammonis sclerotic, shrunken and hard. Separated by Meynert's method, right hemisphere weighed twenty ounces; left, fourteen and a half ounces. Weight of brain, forty-four and a half ounces. No very noticeable difference in corpora striata. Left optic thalamus not much more than half the size of right; harder in consistency. Cerebral substance, except as above mentioned, of normal consistency and color; blood-vessels healthy.

Case 5.—F. S., a woman, aged 46, died in the Danvers Lunatic Hospital, June 29, 1895, in status epilepticus. She was said to have had a "shock" at the age of five years, ever since which time she had been paralyzed in the right side and subject to convulsions.

The hemiplegia was of moderate severity, leaving her some use of the hand. She was imbecile, but could read. Convulsions not very frequent.

At the autopsy the cranium was asymmetrical, the left side being smaller. The anterior part of the falx cerebri was wanting, and the arachnoid of the frontal lobes was adherent on their adjacent surfaces. The left hemisphere was smaller in all its dimensions than the right, especially behind the fissure of Rolando. The posterior third of the corpus callosum was reduced to a translucent membrane. The left lateral ventricle was greatly dilated. In the parietal region, at the bottom of the sulci, the cerebrum was about one-eighth of an inch thick. The entire hemisphere was of firmer consistency than the left and of lower specific gravity, as shown by a tendency to float in the hardening fluid. The left cornu ammonis was not more than half the size of the right and very much harder. The left corpus striatum was decidedly smaller than the right, and the left optic thalamus not more than half the size of its fellow. The brain weighed 1,011 grammes; right hemisphere, 485 grammes; left, 270; right half of cerebellum, 61; left, 62.

Microscopically, the right cornu ammonis is normal in appearance. In the left, the nerve-cells of the nucleus fasciæ dentatæ are almost entirely wanting. The same is true

of the stratum pyramidale with the exception of a small part of its convexity, just before entrance into the fascia dentata. The stratum granulosum is well preserved. The neuroglia of the atrophied portions contains numerous nuclei, but no large spider cells. In specimens stained by Pal's method the deep layer of the alveus and its radiations into the nucleus fasciæ dentatæ are inconspicuous. In the remainder of the cerebrum there seems to be some thickening of the superficial layer of neuroglia in both hemispheres. Otherwise, the right hemisphere seems normal. In the left there is everywhere a diminution of the thickness of the third layer of nerve-cells, and of the number of large pyramidal cells in this layer. The large cells peculiar to the excitable region are well developed. Medullary stains do not bring out any very striking difference in the number or size of the fibres in a given area.

Case 6.—E. P., male, aged 21, died in Danvers Lunatic Hospital Dec. 27, 1895, of diarrhœa. No history of his disease previous to admission was obtained. The right side of the face and right extremities were smaller than the left. There were no contractures, on the contrary, the joints of the right fingers were abnormally loose. There were athetoid movements of right hand. He was almost completely idiotic and talked very imperfectly. He was subject to fits, usually nocturnal, in which he would stand and whirl about, but did not fall.

At the autopsy, after removing the calvarium and dura, there was little difference apparent in the size of the hemispheres, but the left fluctuated when handled. On taking out the brain, the left hemisphere collapsed, a large amount of fluid escaping. The left lateral ventricle was greatly dilated, especially in its posterior part. The entire occipital lobe was very much thinner; over most of the external surface it was only a translucent membrane. The frontal and parietal lobes of this hemisphere were of normal consistency, but the temporal was indurated throughout. Both cornua ammonis were small and indurated. No other localized lesion of the right hemisphere was noticed, but, although symmetrical, it was small, weighing 369 grammes against 187 for the left. The right half of the cerebellum weighed 42 grammes; the left, 58. The entire weight of the brain was 743 grammes. The left optic thalamus and corpus striatum were smaller than the right. In this case, if the left hemisphere had weighed as

much as the right, it would have brought the total weight of the brain to only 925 grammes—a distinctly microcephalic weight. In view of this fact, and of the condition of the cornu ammonis, it seems probable that both hemispheres were affected, in this case, by the morbid process.

Microscopically, in both cornua ammonis, there is absence of most of the large nerve-cells of the nucleus fasciæ dentatæ and stratum pyramidale. Stratum granulosum well preserved. Nuclei of neuroglia in atrophoid portions numerous, but spider cells absent. The left gyrus hippocampi, in a portion adjacent to the cornu, is similarly atrophied in its whole thickness. Medullary staining shows the deep layer of the alveus and its radiations into the fascia dentata to be almost absent in the right cornu and greatly thinned in the left.

In the foregoing six cases of infantile hemiplegia, the essential nature of the lesion was evidently the same in all cases, although the reduction in size of the hemisphere took place sometimes more on the outer, at others on the inner surface. It seems to me altogether most probable that the lesion of the cornu ammonis of the affected hemispheres was due to the same cause, whatever that may have been, as that of the other convolutions, rather than that it was an independent change. In the remainder of the hemispheres the appearances varied too greatly for concise description. Where there was complete destruction of the nerve-cells of the cortex, the histological appearances were identical with those of the diseased parts of the cornua.

To be continued.

STUDY OF THE HANDS IN ACROMEGALY BY MEANS OF THE ROENTGEN RAYS. G. P. Marinesco. *Comptes Rendues de la Société de Biologie.* June, 1896.

The author records four cases which he observed, one apparently of the "giant" type and the others probably of the thick set or "clumsy" type. In the first there was no new osseous formation of the bones at the extremities. The others showed some hypertrophy of the epiphyses, therefore the conclusion was reached that in the "clumsy" type the hypertrophy of the soft parts of the bones is greater than in the "giant" type.

In a case of erythromelalgia which also came under the author's observation, no skeletal changes were to be noted, thus showing that it was not to be classed with acromegaly.

JELLIFFE.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, January 5, 1897. B. Sachs, M. D., President.

ACUTE POLIOMYELITIS WITH INVOLVEMENT OF THE SEVENTH NERVE.

Dr. Terriberry presented a child, twenty months old, exhibiting this condition. This was the second one, and was born of healthy parents after a normal gestation and confinement. It was breast-fed and developed normally. It walked without support at the age of seventeen months. It had never received any injury. On November 30, 1896, the child was noticed to be restless and irritable, and the following morning there was high fever with constant nausea and a profound prostration of the muscular system. The child appeared to be entirely free from pain and tenderness. When it was held up, the head fell on the left shoulder. Twenty-four hours later the condition was not so severe. At this time the right side of the face was noticed to be unnatural, the eye being constantly open. It was not until the sixth day that the child was able to balance itself on its feet. He had first seen the child ten days after the onset of the attack. Examination then showed complete Bell's paralysis on the right side of the face. The right foot pointed outward and there was a slight limp on this side. The knee-jerks were about normal. The galvanic current that could be tolerated produced good reaction on the unaffected side, but on the right side there was very slight reaction and the formula was normal. Since that time the galvanic current had been applied two or three times a week. At the present time the eye was better and the limp was scarcely noticeable. There was slight flabbiness of all the muscles on the right side. The clinical picture presented by this case was quite typical of an acute poliomyelitis.

Dr. Joseph Collins thought it was rather hazardous to make a diagnosis of anterior poliomyelitis in an infant who a month ago had the beginning of such a destructive lesion. A much more probable interpretation would be an infection of the central nervous system, manifesting itself not only in the spinal cord, but in other parts of the nervous system, this infection not being sufficient to cause death of the ganglion

cells. A fully developed anterior poliomyelitis was not compatible with the degree of improvement exhibited in this case.

Dr. William B. Noyes called attention to a class of cases which had been very prevalent this fall, beginning with symptoms almost exactly like poliomyelitis and characterized by unusually marked sensory disturbances and recovering after three or four weeks. He could recall seeing since September about six such cases at the Vanderbilt clinic. In these, the peripheral nerves were sometimes affected at the same time as the spinal cord. This would, perhaps, offer an explanation of the case just presented.

Dr. Terriberry said that in a neuritis of a mixed nerve one expected to find some sensory disturbance. There was entire absence of sensory disturbance in the case presented. He knew of no category in which the case could be placed except in that given—*anterior poliomyelitis*. The theory, of course, mentioned by Dr. Collins we all recognized. Owing to the clinical features of the case he thought he was justified in so classifying it.

Dr. Collins said that he should classify the case as a *bulbar myelitis*. He believed pathologists stated now that the entire gray matter might be affected at the same time. It appeared to him to be a step backward to look upon the case as one of *anterior poliomyelitis* with bulbar involvement.

GRAVES' DISEASE WITH ŒDEMA OF THE EYELIDS.

Dr. Scratchley, on behalf of Dr. Bishop, presented a lady suffering from Graves' disease. This condition had lasted for four years and a half. The case was presented because of the *œdema* of the eyes, which had become much more manifest during the past few weeks. Various methods of treatment had been tried, but the best results had followed the use of *belladonna*.

Dr. J. Arthur Booth said that the kind of *œdema* found in this case was not uncommon when the eyes were very prominent. In the case which he had reported previously there had been marked *œdema* without prominence of the eyes. At the present time he was experimenting with *thymus extract* in these cases.

COMPRESSION PARAPLEGIA IN POTT'S DISEASE OF THE SPINE, BASED UPON STATISTICS.

Dr. V. P. Gibney read a paper with this title (see this journal, page 195).

Dr. Newton M. Shaffer presented a paper entitled

A CONSIDERATION OF THE PARAPLEGIA OF POTT'S DISEASE
WITH ESPECIAL REFERENCE TO THE RESULTS
OF MECHANICAL TREATMENT.

(See page 210).

Dr. De Forrest Willard, of Philadelphia, presented skiagraphs of two cases now under his care and read a paper entitled

LAMINECTOMY IN SPINAL CARIES PARAPLEGIA.

(See page 223).

Dr. Robert Abbe said he had felt for a long time that the cases upon which the surgeon should operate were those which the orthopedic surgeon gave up, and these were very few. The surgeon was reluctant to operate because of the danger of disseminating tuberculosis by the operation. He firmly believed that if Nature had an opportunity she would surround the tubercle bacilli with a fibrous barrier which would localize the disease and prevent general infection. This doctrine of avoiding resection of tubercular parts applied with especial force to the spine. Simple laminectomy was not especially difficult, although it was followed by great shock. He believed that the U-shaped incision and the free division of the muscles were responsible for much of the hemorrhage and shock. By simply incising along side of the spine down to the laminæ no muscles were cut through. The muscles are scraped away from one side, and the laminæ with attached muscles are drawn to one side. The laminæ could then be divided without undue hemorrhage. By this method the operation was extremely safe. If there were much handling of the cord undoubtedly there would be great shock. By picking up the dura he had been able to roll the cord to one side, so that he could inspect its anterior surface without producing shock. There was, of course, much unavoidable manipulation if one would thoroughly remove the disease and cure the patient. The laminæ are not at all necessary to the support of the spine, but they may be missed if the bodies of the vertebræ are extensively destroyed. Even after primary union, a sinus is apt to form after a short time, owing to imperfect removal of the disease, and this sinus interferes with mechanical appliances.

Dr. Joseph Collins said that he had been taught that one of the most important diagnostic signs of compression para-

plegia was an increase of the reflexes, and, although he believed that this was the rule, yet in his own experience there was a number of cases with absence of the knee-jerk and without ankle clonus. He had, with Dr. Fraenkel, seen no less than seven cases, at least three of which were cases of Pott's, in which these conditions were noted. This condition of the reflexes is not easily explained, but it seemed to the speaker that the theory propounded by Bastian in 1879 and since elaborated by him, allowed of a more satisfactory interpretation than did any other explanation that had been suggested. In fact, the theory founded on the cut off influence existing between the reflex arc and the cerebellum seemed to have the greatest support, but he thought that it would yet be found that the condition of the reflexes in transverse lesions of the cord would be just in proportion as the columns of Gowers were affected or spared. The report just made by Dr. Gibney of nearly 50 per cent. of recoveries should be sufficient evidence to convince any one that there is very little justification in subjecting a patient to an operation that is so hazardous as Dr. Willard has told us laminectomy is. The degree of deformity he had found to bear very little relation to the extent of paralysis. He recalled a case in which there was no evidence of disease of the spine from ocular or manual examination, yet the paralysis was very great. He had observed absence of menstruation in one case and recalled that he had spoken of this case before the Orthopedic Section two years before. Iodide of potassium he believed was useful in these cases, yet the use of this remedy, to the exclusion of mechanical treatment was to be deplored.

Dr. M. Allen Starr said that from experience with operations on the spinal cord for other conditions, he thought we should be very cautious about meddling with the cord, and that if the cord had been once damaged it was practically useless to place it in a favorable condition for recovery, as it did not seem to have the power of regeneration. He had been surprised at the remarkable improvement that had occurred in cases of almost total paraplegia as a result of careful mechanical treatment. It should make us careful in the prognosis given in all cases of compression paraplegia. It was very rare for him to see a case in which the tendon reflexes were not very markedly exaggerated, so that his experience did not at all coincide with that of Dr. Collins. The majority of observers, he believed, still continued to find the knee-jerks exaggerated and clonus present when the cord was compressed above the mid-dorsal region.

Dr. C. A. Herter said that the pathological conditions underlying the desperate cases of compression myelitis were such as to give very little hope of anything more than the most

temporary relief from operation. At the same time, he would regret exceedingly if all the surgeons took the stand taken by Dr. Abbe, for there was now enough reliable evidence to justify the continuance of this operation in the very severe cases. He had not met with loss of tendon reflexes in compression myelitis where the lesion was below the mid-dorsal region. He had only observed alternation of increased reflexes and loss of reflexes in cerebellar disease and in some cases of meningitis. The term "compression myelitis" seemed to him perfectly proper, as describing an effort at repair on the part of the cord. In his experience the compression had been due rather to the accumulation of broken-down tuberculous material outside of the dura than to the bone itself. The softening of the bone appeared to be due to tubercular disease of the vessels, similar to that found in patches of syphilis of the cord.

Dr. Edward D. Fisher said that he agreed with those who recognized exaggeration of the reflexes as a symptom of approaching compression paraplegia. Occasional variations should be looked upon merely as individual peculiarities. It was generally conceded that the cause of the compression was the infiltration between the bone and the dura, and hence it seemed to him that there was some indication for an operation if this material were not absorbed after a reasonable time. The question of operation should be decided by the condition of the patient rather than by the length of time the paralysis had lasted—in other words, by the acuteness of the process. He did not think the shock following the operation was due to the handling of the cord as much as to the excessive hemorrhage. The cord could be handled without giving rise to immediate shock, and it was difficult to see why shock should follow handling the cord in the lower dorsal region. In a large number of cases it was the surgeon's duty to operate.

The President said that the question of operation came up only in desperate cases, and in those in which there was a great probability that the paralysis would continue for the remainder of the patient's lifetime. It had been intimated that this was a condition which did not ordinarily tend to a fatal termination, but, in his opinion, a very considerable proportion did die from the disease. It was, therefore, the duty of the surgeon to make the percentage of recoveries as large as possible. He believed, with Dr. Abbé, that the question of shock was largely one of hemorrhage, and hence was probably preventable by improved technique. Certainly the question of shock in cranial surgery was chiefly one of hemorrhage. It was the duty of the neurologist to determine at what time the operation should be done. Where bone disease threatened extensive involvement of the spinal cord the operation would hardly be indicated, because the spinal cord would have been

already damaged beyond repair. For this reason, if the neurologist had reason to believe that the cord was liable to be extensively destroyed, he should advise operation at once. He recalled a case of Pott's disease in which the first symptom was a peculiar sensory disturbance, which preceded the kyphosis by a number of months. Again, the symptoms often point to a compression of the lateral and posterior portions of the cord. It would seem that the performance of laminectomy at this period might avert further destruction of the cord. He had not seen a single case of Pott's disease in which the knee-jerks were lost; on the other hand, he had uniformly found a marked exaggeration of the reflexes.

Dr. Shaffer, in closing the discussion, said that out of his forty cases there were only four that did not show some indication of recovery and the great majority of cases recovered within one year. He, therefore, believed that the prospects of mechanical treatment were much greater than could be held out by any other method of treatment.

DIE DIFFERENTIALDIAGNOSE DES HIRNABSCESSSES. [The Differential Diagnosis of Cerebral Abscess.] Berliner klinische Wochenschrift, Nos. 45 and 46. By H. Oppenheim.

The diagnosis of cerebral abscess is not always easy. One may be called upon to decide between abscess and meningitis due to trauma. The cerebral hemorrhage, which does not develop until some time after an injury (Bollinger), may present many similar symptoms. Trauma, or erysipelas resulting from a wound of the head, may cause non-purulent encephalitis, but they may also cause abscess. Functional neurosis may give rise to many difficult questions. While the symptoms of irritation and paralysis in these neuroses are usually found on the same side of the body as the cranial lesion, abscess, produced in the opposite cerebral hemisphere by *contre-coup*, may give rise to a similar location of symptoms. Friedmann has found that certain changes in the smaller cerebral vessels may cause symptoms, developing after trauma, which closely resemble those produced by abscess. There are a few cases of so-called abscess in which no organic changes have been found. Circumscribed purulent meningitis may clinically resemble cerebral abscess, and in some cases may be associated with it. Meningitis resulting from otitis may be limited to the spinal membranes. Fever is not always observed in cerebral abscess; slight and temporary elevations of temperature are not infrequent, but continuous and high fever renders the diagnosis of uncomplicated abscess improbable.

The slowing of the pulse is one of the most valuable diagnostic signs of abscess. Lumbar puncture is dangerous in a case of suspected abscess, as the sudden diminution of pressure may cause discharge of the pus. Uncomplicated otitis may stimulate cerebral abscess. Peptonuria may be found in encephalitis as well as in abscess. SPILLER.

Periscope.

With the Assistance of the Following Collaborators:

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THE DEVELOPMENT OF THE BRANCHES OF THE FIFTH CRANIAL NERVE IN MAN. A. Francis Dixon, B.A., M.B., Tr. Royal Dublin Society. 1896.

From the study of human embryos of the fourth to eighth week, the author reaches the following conclusions relative to the development of the fifth nerve:

Three divisions arise from a single undivided Gasserian ganglion: Before the origin of the ophthalmic a cellular cord stretches from the ganglion and occupies the place of the future nerve cord. From the ganglion axis cylinder processes grow out into this cellular cord; in this manner the ophthalmic branch is formed; by the sixth week all of the ophthalmic branches are represented. The ciliary ganglion is present at this time and has the same relations as in the adult. It is not a homologue of a spinal ganglion. In the sixth week the otic ganglion and Meckel's ganglion are in close connection. The chorda tympani and vidian branches do not arise from the fifth nerve, nor are the cells of the accessory ganglion derived from the Gasserian.

JELLIFFE.

SUR UNE PARTICULARITÉ DE STRUCTURE DES CELLULES DE LA COLONNE DE CLARKE ET SUR L'ÉTAT DE CES CELLULES DANS LE TABES SIMPLE OU ASSOCIÉ À LA PARALYSIE GÉNÉRALE. [Concerning a Peculiarity of Structure of the Cells of the Column of Clarke, and the Condition of these Cells in Simple Tabes, or Tabes associated with General Paralysis.] *Revue Neurologique*, Nov. 21, 1896. By G. Marinesco.

Marinesco has examined three spinal cords from patients afflicted with general paralysis and tabes. In general paralysis the cells of the column of Clarke are tumefied, and the nucleus in most of them is displaced to the periphery. The chromatophilic elements form a thin layer along a portion, or the whole of the border of the cell. The nucleus is seldom round, more commonly is elliptical and flattened, occasionally reniform or crescentic. The nucleus may cause a rupture of the cell wall, and in this way form hernia. The more eccentric the nucleus is, the fewer are the chromatophilic elements. The processes of the cells are less numerous. In simple tabes the cells of the column of Clarke show less intense lesions. The cells in the posterior cornua are also in part altered.

In the cases examined the cells of the anterior cornua were not all intact, and Marinesco believes the alteration in these is more common than is usually believed.

In normal cells the nuclei are central, for the centre of the cell represents the region of maximum nutrition.

Marinesco found some alterations in the cells of the column of Clarke in patients who had not had tabes or general paralysis, but these changes were always of less grade. SPILLER.

ON THE GROUPING OF THE ENDOGENIC FIBRES IN THE POSTERIOR COLUMNS OF THE CORD. [Sur le Groupement des Fibres Endogènes de la Moelle dans les Cordons Postérieures.] H. Dufour. Archives de Neurologie, Aug., 1896.

The author, after reviewing the contradictory opinions of a number of observers as to the presence of descending fibres in the posterior columns, gives the result of his studies upon a case in which the posterior roots were involved by an epitheliomatous mass from the third lumbar nerve downward, the disease having been present about five years. The cord itself was not macroscopically involved.

The posterior roots were studied. Those of the sacral region were entirely destroyed; those of the lumbar still contained some healthy fibres.

The author comes to the conclusions which he contends are in accord with recent observations by Souques and Marines (Presse Medicale, 1896; and Hoche, Neur. Cent., 1896), that there is a tract of fibres in the posterior columns occupying a central position that degenerates downwards. These fibres are not to be confounded with descending collaterals of the posterior root fibres.

The author sums up his observations by the following conclusions:—

(1) The presence of descending collateral fibres is not yet absolutely demonstrated for the adult, but even if it be granted that they are present, this would not negative the presence of endogenous fibres suffering descending degeneration in the posterior columns of the cord.

(2) Two sets of fibres may be demonstrated, called endogenous or commissural. One anterior set forms a short tract, the other posterior, forms, with some of the fibres at least, a long tract. JELLIFFE.

D'UN FAISCEAU SPECIAL DE LA ZONE LATÉRALE DE LA MOELLE ÉPINIÈRE. [Concerning a Special Bundle of Fibres in the Lateral Zone of the Spinal Cord.] Revue Neurologique, No. 23, 1896. By A. Bruce.

The researches of Cajal have shown that the lateral limiting layer, situated close to the gray matter between the anterior and posterior horns, consists of short fibres which arise and terminate within these horns. Bruce has examined a case of amyotrophic lateral sclerosis, in which not only the pyramidal tracts but also the greater part of the antero-lateral columns, with the exception of the ascending tracts, was degenerated. The anterior part of the lateral limiting layers was also affected, while the posterior was deeply stained. It would appear that the degenerated fibres in the former arose in the anterior horns, and the normal fibres of the latter portion chiefly in the posterior. It is necessary, therefore, to speak of the ventro-lateral and the dorso-lateral limiting layers. SPILLER.

ON THE PHENOMENA OF REPAIR IN NERVE CENTRES AFTER SECTION OF PERIPHERAL NERVES.

M. G. Marinesco (La Médecine Moderne, Nov. 18, 1896) reports some interesting observation upon this process. Experience has shown

that after the section of a nerve a certain effect is produced upon its centres, but we are almost wholly ignorant of the phenomena which take place during the process of restitution to health. M. in studying this question, cut the hypoglossal nerve in several rabbits, and found that if nothing interfered with the reunion of the cut ends, the phenomena of repair in the nerve cells were very marked at the end of twenty-four days. The nucleus of a cell taken from a cut nerve could readily be distinguished under the microscope from a corresponding nucleus of the intact hypoglossal nerve. Two modifications were especially noted in the cells; one an increase in size, the other a more intense coloration as the result of an overgrowth of chromatophilic elements. These often showed this in the form of filaments wholly occupying the entire area of protoplasm, in others disposed about the nucleus. Sometime a clear zone was interposed between the peripheral and peri-nuclear chromatophilic elements. At the end of ninety days hypertrophy of the cells continues to be evident. During all these periods of repair the number of cells in the normal nucleus is larger than that of the nucleus in the course of restoration, making it evident that the cells of the latter make up in volume what they lose in number.

MITCHELL.

UEBER HIRNABSCESS NACH STIRNHÖHLENEITERUNG. [Cerebral Abscess following Suppuration in the Frontal Sinuses.] Deutsche medicinische Wochenschrift, Nov. 19, 1896. By Treitel.

Dr. Treitel calls attention to the fact that cerebral abscess following disease of the nose, and especially of the frontal sinuses, has not been frequently mentioned. He reports a case in detail in which the sinuses were opened on account of a purulent nasal discharge. After a time convulsions and right hemiparesis were noticed, and the diagnosis of abscess near the left central gyri was made. Repeated punctures of the brain failed to reveal any pus. At the autopsy purulent basal meningitis and abscess of the left frontal lobe were found. It was believed that at first there were small multiple abscesses, and that, for this reason, no pus had been obtained. Had the punctures been made after the abscesses had united, pus might have been found.

Usually in suppuration within the frontal sinuses the posterior wall becomes carious, and the pus in this way passes to the brain. The advice is given to remove the anterior wall of the sinuses in every case, as cerebral complication is very common in disease of the sinuses and may be latent. Treatment by this procedure is more efficacious, and the operation does not produce disfigurement.

SPILLER.

SYPHILIS OF THE NERVOUS SYSTEM.

The following cases are reported by McCall Anderson in a lecture on the less common forms of syphilitic disease. (Brit. Med. Jour., Sept. 12, 1896.)

On January 16, 1896, a miner, aged 39, was admitted into the Western Infirmary complaining of headache and of fits. The family and previous personal history were good. In May, 1895, while wrestling in a public house, he fell, striking his head against the floor. Thereafter he suffered greatly from headaches, which were not localized, and six weeks after the injury he had his first fit, there having been eight in all, occurring at intervals of three or four weeks. The earlier seizures were preceded by an aura sufficiently prolonged to allow of his lying down, and the first one was associated with complete loss of speech for three days. In the first two paroxysms the convulsions were general, but the three following were confined to the right side of the body and face. The last three seizures were unaccompanied by loss of consciousness, and were limited to the arm, while there was only a numb feeling in the face. For a couple of weeks before admis-

sion he experienced a loss of power on the right side, especially in the arm. Occasionally, when he walked about, there was some vertigo, the headache persisted, and he had a difficulty in reading, for though he could see the words and say them, he was hardly able to follow the sense, but there was no loss of memory.

On admission it was observed that his expression was heavy and stupid; he spoke slowly and indistinctly, as if he had not only difficulty in getting words to express his ideas, but also in articulating them. There was paresis of the lower part of the right side of the face, and, to a less extent, of the corresponding arm, but not of the leg; also some rigidity of the right elbow, and more marked stiffness of the knee. Sensation was slightly impaired as far as the sense of pain and of temperature were concerned, and all the deep reflexes were exaggerated on this side, while ankle clonus was present on both sides. The superficial reflexes were normal, and the special senses were not impaired. There was well-marked tenderness over a limited area above and behind the left ear.

Because of the nocturnal character of the headache, extensive scars on penis, a "tissue paper" scar on the leg and enlarged glands, a diagnosis of cerebral gumma was made, and under specific treatment the patient entirely recovered.

The next case we would not consider very unusual. A gentleman, age 38, noticed difficulty in pronouncing certain words; three days later he was for a short time unable to speak. For ten days thereafter he experienced occasionally a numbness of the left cheek and point of the tongue, and a distinctly metallic taste. On the evening of the 16th, he suddenly suffered from a choking sensation, followed by contraction of the left side of the face, and loss of speech. He was quite intelligent at the time, but felt some confusion of mind. The power of speech returned in about an hour, but from the 17th to the 31st he became speechless four or five times. During all this time he was unable to make the slightest calculations, and could not spell words correctly, with the single exception of his own name. When I saw him on August 31st, there was a little permanent aphasia; he could not write very accurately (for example, for Fairlie he wrote Fairlia, although he knew that he was making a mistake), and he was suffering from severe pain, chiefly in the occipital region, which set in some time after the onset of the aphasia.

These symptoms were supposed to have a syphilitic basis for the following reasons:

1. Thirteen years before he had a solitary chancre on the penis.
2. The pain in the head was nocturnal in character.
3. There were coppery stains on the legs and on one arm.
4. There was an ulcerated patch on one leg, the edges of which were perpendicular; it was circular in shape, and the skin around had a coppery color.

Treatment consisted of the inunction of Shoemaker's mercurous oleate ointment (3j daily). For the first two nights the pain in the head was worse, but in a very few days it disappeared, and improvement in pronunciation and power of speaking set in. In three weeks all the symptoms had disappeared.

The author reports the following as a case of locomotor ataxia, "in its initial stage," cured by specific treatment. It is palpably a case of active syphilis of the cord—pseudo-tabes syphilitica.

A gentleman, aged 43, came to me on February 3d, 1885, suffering from symptoms of four weeks' duration. These consisted of staggering gait, with numbness in the lower extremities, and inability to feel the ground properly when walking. For a week, too, he had experienced numbness in the arms (especially the right) up to the elbows, and limited to the region of distribution of the ulnar nerves (the thumb, index, and corresponding side of the middle fingers escaping).

His knees felt slightly stiff, and he suffered from a feeling of tightness across the stomach and of much flatulence, especially after food, and at times he had a feeling as of a band across the abdomen. The knee jerks were absent. He attributed his illness to going out after sitting in a hot office; but, ten years before I saw him, he had a single chancre on the penis, followed by blotches on the skin and pains in the bones, which, however, did not trouble him long, and for which he was only treated for a short time.

Treatment was commenced on February 4, a drachm of mercurial ointment being rubbed into the skin daily. By the 7th the numbness of the legs had disappeared, while that of the arms was less pronounced. On that day the ointment was increased to two drachms, and he was directed to apply galvanism to the spine for ten minutes daily (ten cells of a Leclanché battery).

On February 19th the following note was taken: "The numbness in the arms has been steadily diminishing, and he now walks well." At the end of March he went to Jersey for a change of air, and, although the weather was bitterly cold, he continued to improve, so much so that when he returned on April 28th he was quite well, a little numbness of the right arm alone remaining. He remains well to this day.

The last case is certainly noteworthy because of the completeness of recovery from such severe symptoms.

A timekeeper, aged 42, was admitted on January 8th, 1894, suffering from loss of power and numbness of the lower extremities, with severe pain in the legs below the knees, of three weeks' duration. He stated that he had all along been very healthy, with the exception of an attack of pleurodynia of a few days' duration in 1889, and he had always been very temperate. For some weeks before the onset of the threatening symptoms he had a severe cold, which he neglected. But about three weeks before I saw him it became worse, and he had to take to bed, when he was found to be suffering from pneumonia. Two days thereafter he began to suffer from pain in the legs, and the following day, on attempting to get out of bed, he fell at full length upon the floor. He was lifted into bed, and then it was found that the lower extremities were completely paralyzed, and he complained of numbness in them. Soon after this his bladder began to trouble him; he had difficulty of micturition, sometimes amounting to retention, and pain across the epigastrium. His bowels were constive at first, but they soon became loose, and then he lost control over sphincter and passed all his movements in bed. About a week after the onset of the symptoms several bedsores formed over the sacral region.

On examination it was found that the lower extremities were absolutely paralyzed, only a slight degree of movement of the toes being possible. Anæsthesia was absolute and extended nearly to the umbilicus. He had a sensation of tingling in the feet, and to a less extent in the hands also. The knee jerks were quite absent. He said that there was no loss of power in the arms, but the dynamometer registered only 10 kilos in either hand. The bladder was overdistended, and the urine was therefore removed by catheter. The pains in the legs were constant and severe, but more intense at some times than at others: they were worse in the feet, but had no shooting character.

I suspected that these symptoms were due to syphilis, for the following reasons:

1. Twenty years ago, after exposure to infection, he had a "very trifling" affection of the penis, the precise nature of which he could not define, and for which he was treated by internal remedies for a fortnight only and "cured." No secondary manifestations were noticed.

2. The pains in the legs were markedly worse at night, and he also had occasionally slight nocturnal headache.

3. He married in 1881 at the age of 30. The following record gives the issue of his wife's pregnancies:

- (a) Six months' child, stillborn, March, 1882.
- (b) Seven months' child, stillborn; rash on trunk.
- (c) Eight months' child; said to have been dead for two weeks before birth.
- (d) Six months' child, stillborn.
- (e) Female child, at full time, apparently healthy; is now 5 years old, and is in the Royal Infirmary with disease of the ulna and tibia.
- (f) Miscarriage at two or three months.
- (g) Male child, apparently healthy; died a fortnight after birth of "collapse of lungs."
- (h) Miscarriage at second month in 1893.

The condition of the patient, then, before treatment, was as follows: Paraplegia absolute (only slight movement of toes), anæsthesia complete, nearly up to umbilicus. Constant and severe pains in the legs and feet; absence of knee jerks; paresis of the upper extremities; retention of urine; incontinence of fæces; four large, deep bedsores over the sacral region, so that it would be difficult to find a more unpromising case for treatment.

He was put upon a water bed, the bladder and bowels were attended to, and the bedsores dressed with boracic powder. Daily inunction of 3j of mercurial ointment was commenced on January 9th, and for some time antipyrin (gr. xv.) was given every evening on account of the pain in the legs.

A fortnight thereafter he could draw up his legs in bed, and every day further recovery was observed: the anæsthesia also was less marked, and gradually disappeared. By January 28th the retention of urine and incontinence of fæces had passed away, and the antipyrin was stopped because the pains in the legs were gone.

The bedsores healed rapidly, and were quite cicatrized about the same time. On February 10th he was able to rise and walk a short distance in the ward, and after time massage was added to the other treatment. He could walk a longer distance every day, although his legs were tremulous at first. By February 28th the grasp of the hands was much stronger, the dynamometer registering 36 kilos in the right and 30 in the left hand, as compared with 10 kilos on admission. He left the infirmary on March 13th, and before leaving he was shown at the meeting of Glasgow Pathological and Clinical Society. His recovery was perfect. He could walk as well as ever he did, and the knee jerks even had returned.

PATRICK.

A CASE OF CEREBROSPINAL SYPHILIS. *Deutsche Zeitschrift für Nervenheilkunde*, Vol. IX., Nos. 1 and 2, 1896. By Richard Cassirer.

A case of right hemiplegia with oculo-pupillary symptoms, headache and mental disturbance was shown by the autopsy to be due to cerebrospinal syphilis.

Intense degeneration of the intramedullary portion of the left trochlearis was one of the most interesting lesions found. Affection of this nerve has not been often noticed in cerebrospinal syphilis, according to Cassirer. The spinal root of the trigeminus ("ascending root") was degenerated. This was probably the result of meningitis. The fibres of this nerve at the point of entrance, as well as the motor and sensory nuclei appeared intact. The tendency of basal meningitis to involve certain fibres of a nerve to the exclusion of others—not infrequently observed in the syphilitic affections of the oculomotorius—was thus shown in the trigeminus. A lesion in the Gasserian ganglion, however, was not excluded.

When in tabes the spinal root of the fifth nerve is degenerated, the affection is usually bilateral.

SPILLER.

Book Reviews.

TRAVAUX DE NEUROLOGIE CHIRURGICALE. 1895. By A. Chipault, Braquehay, Demoulin, Daleine. Paris: Battaille & Co. 1896.

This work on the surgery of the nervous system is not complete, and is intended to be the first of a series by the same authors. The studies are largely based upon the authors' personal observations, and the book is therefore more valuable than a compiled manual. Particularly interesting are the chapters upon the intra-dural resection of the posterior roots, upon Quincke's puncture, and a series of very interesting cases, by Chipault himself, upon lesions of the brachial plexus from fractures of the clavicle and the surgical treatment thereof. In the first of these C. gives full credit to Abbe's suggestion of this operation, which was indeed made in the same week that W. H. Bennett proposed it in London. The seven cases which had been operated upon up to the publication of the present study are all considered in detail. In only four of them, one of Abbe's, one of Horsley's, and two operated by Chipault and Demoulin was the operation, properly speaking, an intra-dural resection. It may be quoted as especially interesting that in the first case upon which the authors operated, one of extreme hyperæsthesia of the right fore-arm and hand the cedema and ulcers which had covered the ulnar side of the arm and fingers, disappeared in twenty-four hours after the section of the seventh and eighth cervical and first dorsal roots, and the patient made an otherwise excellent recovery.

A dispassionate and judicious review of the dangers and advantages of vertebral punctures precedes a report of five personal cases.

The chapter on fracture-lesions of the plexus contains reports of seven personal cases, and a very interesting discussion on the neural symptoms from which it may be judged that injury of the plexus has taken place. The discussion of treatment, preventive and curative, follows, and the author concludes as follows:

"The unfortunate consequences of lesions of the plexus from clavicular fracture can almost always be cured or greatly lessened by active surgical treatment, provided it is not too late, and provided it is properly done. It appears to us proper, in spite of the possibility of spontaneous amelioration in slight cases, always to interfere when the diagnosis is once made, and, in order that therapeutic conditions may be the best, to interfere at once and thoroughly: that is to say, to make a resection of the injured portion of the clavicle, followed by osseous suture."

MITCHELL.

ATLAS DER PATHOLOGISCHEN HISTOLOGIE DES NERVENSYSTEMS.
[L'Anatomie Fine de la Moelle Epinière.] Berlin, 1895. Herausgegeben von Babes, etc. iv. Lieferung.

S. R. Cajal gives a short account of the normal histology of the spinal cord, with eight plates. An atlas cannot be expected to go into all the details, but even with this excuse, the presentation is a disappointment, surely, as far as the part of the work is concerned which we do not owe to Cajal exclusively. What we need most is a

plain statement of what is to be considered a fair standard of the normal, especially at the various stages at which specimens usually reach the pathologist and with the methods in current use. Instead of that, we are treated to an embryological study, and a very defective statement of what we see with the Weigert stain.

"Inspired by the character of the publication in which this work appears, we shall restrict ourselves to present by the means of drawings, copied as exactly as possible from nature, the facts of the structure of the spinal cord which seem to us best established and easiest to control." If this were taken in the sense spoken of above, Cajal might have given us more than a poor résumé of what every recent text book, or his older publications, give us fully as well.

After a short summary of the kinds of cells and fibres, and the principal generalities about the neuron, the first plate is explained, half a cross section of the spinal cord of a mouse, with the cell-bodies pink, their axons red, the collaterals black, and final arborizations green.

Plate II. is a study of the collaterals in the chick of 15 days' incubation, Plate III. gives the types of cells in a chick of 18 days, Plate IV. the substance of Rolando on the 10th day of incubation, Plate V. the same on the 19th day. On Plate VI. we find represented the neuroglia and nerve elements of the new-born dog. With Plate VII. we might come nearest the task of a normal histology included in an atlas of pathology. It gives a Weigert-Pal section of the cervical cord of the adult. It is difficult to realize how this drawing got into the atlas as a prototype of a normal cord section. The distribution of the cells hardly seems typical, and the distribution of the fibres in the white and gray matter is impossible, absolutely schematic—by mistake called, "*copié aussi exactement que possible d'après nature!*" The distribution of thin and thick fibres, so easily made out in any decent preparation is absolutely ignored. The silence with which recent editions of text books pass over these points, is really remarkable. The study of marked degeneration and Golgi specimens seems to make anatomists overlook the number of things that can be seen in the normal adult cord with perfect ease.

With Plate VIII. we return again to the new-born mice and the chick for the study of the spinal and sympathetic ganglia.

That this volume should be so disappointing, in spite of the fact that it comes from the hands of the man to whom we are so deeply indebted for research work, might be suggestive of the hint that the student of pathology of the nervous system must make his own studies of the normal, and must know his problems better than the embryologist. It seems that there would be ample space in the literature for a normal histology applicable to the needs of the pathologist.

ADOLPH MEYER.

MONATSSCHRIFT FÜR PSYCHIATRIE UND NEUROLOGIE. Band I. Heft 1. Edited by Prof. C. Wernicke and Prof. Th. Ziehen.

The *Monatsschrift für Psychiatrie und Neurologie* takes at once first rank among the neurological journals of the world. There is an introduction of a few pages devoted to some of the questions of the day. In this attention is paid especially to Flechsig's theory of association centres.

Dr. Karl Bonhoeffer writes a long and carefully prepared paper, entitled, "*Ein Beitrag zur Localisation der choreatischen Bewegungen*" (A Contribution to the Localization of Choreic Movements). He reports a case in which the symptoms were headache, vertigo, choreic movements chiefly in the right extremities and the right side

of the face, ataxia, inability to stand or walk alone, great diminution of the patellar reflex, and paralysis of certain ocular muscles of late development, indicating some lesion near the oculomotor centre. There was no disturbance of motion and sensation, except for the sense of position. Death was due to erysipelas.

The pia was hyperæmic and a mild degree of internal hydrocephalus, probably due to the erysipelas, was noted. A very small carcinoma was found in the right first frontal convolution, which was not supposed to have produced symptoms, and another was found within the corpora quadrigemina. The primary tumor could not be located as a complete autopsy was not permitted. The anterior cerebellar peduncles at the decussation, as well as fibres of the formatio reticularis, were involved in the new growth. The oculomotor paralysis had probably been the result of pressure. The loss of the sense of position and the ataxia were attributed chiefly to the destruction of the fibres of the formatio reticularis, though the cerebellar type of the ataxia was supposed to depend on injury of the anterior cerebellar peduncles.

The most important statements which Bonhoeffer makes after a study of the reported cases of hemichorea are:

1. Hemichorea has not been observed in total hemiplegia.
2. Hemichoreic movements have never been observed as the result of irritation of the degenerating pyramidal tract in hemiplegia of cortical origin.
3. In subcortical lesions of the pyramidal tract hemichorea may only develop when a certain restoration of motor power has taken place.
4. Degeneration of the pyramidal fibres is not necessary for the development of hemichorea.
5. It has not been proven that irritation of the pyramidal tract has in any case produced choreic movements.
6. Lesions of the spinal cord and of the distal part of the oblongata never appear to cause choreic movements.
7. The hemichoreic movements are probably not due to lesion of the motor fibres (Kahler and Pick), but to lesion of a "cerebropetal" tract, as yet unknown, whose function it is to convey impulses necessary for the co-ordination of movement, from deeper portions of the cerebrum to the cortical motor region. It is probable that this tract is to be found within the anterior cerebellar peduncles, for in all cases of chorea which have been caused by focal lesions the fibres of these peduncles have been found injured, and in disease of the cerebellum choreic movements have been observed.

Following the paper by Dr. Bonhoeffer is one by Dr. Hermann Wilbrand, which has the title: "Ueber die Gesichtsfeldmessung am Dunkelperimeter (The Mensuration of the Visual Field with the Dark Perimeter). The visual field is diminished concentrically for a short time when a person is brought into a darkened room, as the entire retina is less sensitive to light after exposure to diffuse daylight, and gradually becomes more sensitive in darkness. Everyone has experienced this dimness of vision in passing from light into darkness. By dark perimeter the author means the instrument used in the darkened room.

"Ueber die Beziehungen der Nervenzellen zu den Neurogliazellen anlässlich des Auffindens einer besonderen Zellform des Kleinhirns (The Relation of the Nerve Cells to the Neuroglia Cells in Reference to the Discovery of a Special Kind of Cell in the Cerebellum) is the title of an important paper from the pen of S. Ramón y Cajal, translated from the *Revista trimestral micrografica*.

If a section of the molecular layer of a rabbit's cerebellum is stained by the method of Nissl, many large, triangular or spindle-shaped cells may be seen in addition to the small stellate cells. These are arranged vertically and have large nuclei and two or three processes containing

chromatin spindles. Numerous nuclei are found about the axis cylinder and lower part of the body of these cells, and are sometimes arranged in the form of a column. The lack of chromophilous protoplasm, the crowding together of these nuclei in a very limited space, which indicates a very delicate cell body, and the reticulated condition of the nucleoplasm cause Cajal to regard them as a variety of neuroglia cells. Similar nuclei are found about many of the pyramidal cells of the cerebrum, about certain spinal cells, about cells of the external geniculate body, and elsewhere. By their arrangement about the non-medullated portion of the axis cylinder where it joins the cell body they may prevent the escape of the nerve current and take the place of the nerve sheath.

Cajal believes that the large nerve cells of the molecular layer are displaced Golgi's cells, i. e., large cells of the granular layer whose cell body has wandered into the molecular layer.

A report of the congress for psychology in Munich is given by Liepmann, and one of the meetings of neurologists and alienists in Frankfurt on the Main, by H. Sachs.

The monthly ends with a notice of the death of the distinguished neurologist, Dr. Carl Eisenlohr, and a few book reviews. SPILLER.

THE DISEASES OF INFANCY AND CHILDHOOD, FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE. By L. Emmett Holt, A.M., M.D., Professor of Diseases of Children in the New York Polyclinic, etc. With 204 illustrations, including seven colored plates. D. Appleton & Co., 1897.

The appearance of this new text-book on diseases of infancy and childhood was well-timed, for, in spite of improved editions and of some excellent qualities, the older text-books on Pediatrics, published in England and in America, had become antiquated.

Dr. Holt's book is nothing if not up to date. His views on infant feeding, on bacteriology, and on pathology are of the latest. Sometimes we fear that the latest authorities have been considered too carefully, while the work of preceding authors has been disregarded. To mention but one instance: On the subject of aphasia, Wyllie's work is quoted, and scarcely another, although it will be conceded that the thoroughly original work in aphasia in children has been done by Kussmaul, Bernhardt, and others, and not by Wyllie; but as long as the student thus gets the benefit of the latest summary, there is not sufficient reason to find fault with the author's methods.

On the whole the book makes an excellent impression: The subject matter is well arranged, the chapters are carefully written, the illustrations are beyond praise, and there is that close relationship between clinical descriptions and pathologico-anatomical observations, which proves that the author has as much respect for the science as for the art of medicine.

Diseases of the nervous system are treated in Section VII., consisting of about 144 pages, and a few in addition are devoted to birth paralyses in the discussion of the diseases of the newly-born. Every author is entitled, in our opinion, to divide up his work as he thinks best, and, granting that Dr. Holt was right in limiting the number of pages to be devoted to nervous diseases, we hasten to add that he has presented the subject, within such limits, carefully and accurately. The author followed in the foot-steps of other writers who have begun with functional diseases and have then passed to the consideration of organic diseases of the brain and spinal cord. In this instance diseases of the spinal cord and of the peripheral nerves are treated after diseases of the brain.

It is very evident in reading these pages in Dr. Holt's book that he has not only consulted freely the authorities on each subject, but that he has had ample opportunities for personal observation, and that his experiences at the postmortem table have been unusually large. It is very evident, however, that his point of view is that of the pædiatrist and not of the neurologist. The general symptoms receive more careful consideration than those special symptoms for which the neurologist is apt to look. Thus, on page 720, in speaking of tuberculous meningitis, the author states that the most important diagnostic symptoms of this disease, enumerated in the order of frequency in 58 cases were as follows: "Obstinate constipation, persistent drowsiness, irregular respiration, vomiting without apparent cause, irregular pulse, convulsions, opisthotonus and fever which was usually slight." Not a word about strabismus, pupillary reflexes and facial palsy! The reviewer has called attention at various times to the peculiar discrepancy between the observations of the general practitioner and the specialist in nervous diseases. We would not attach much importance to this point if practical experience had not taught many of us that physicians have hesitated to make a diagnosis of tuberculous meningitis at a time when the presence of cranial nerve palsies in association with fever, vomiting and convulsions left no doubt in the neurologist's mind that the patient was afflicted with this especial disease.

Several of the chapters on nervous diseases are usually well illustrated, particularly those on meningitis, on hydrocephalus and on cretinism, whereas scant justice has been done in this way to the progressive muscular atrophies. Plate 15, "Acute Meningitis complicating Pleura-pneumonia," is particularly beautiful and true. It would have been better, however, to have described this as acute purulent meningitis, lest the reader should look for such appearances in every case of meningitis, whether or not in association with pneumonia.

There are a few sins of omission which we wish to note in passing. In a book of this description, to which the student would be apt to refer in the first instance, some mention should have been made of the hereditary and family forms of spinal spastic diseases. Multiple sclerosis should have been mentioned, for if it does not occur often in infants, it is apt enough to occur in children of tender age, and needs to be differentiated from other conditions which are more common in early life. It is also true that a few pages might have been devoted to the consideration of syphilis of the nervous system in children, and the chapter on "Progressive Muscular Atrophies"—diseases most common in early life—should have been considered in a somewhat more detailed fashion. We hope that in the next edition of this book a reference will also be made to encephalitis, to migraine, to hereditary cerebellar ataxia in conjunction with the short, and altogether too short, mention of Friedreich's ataxia. It is evident that the author is more interested in infants than in children.

We wish to abstain, however, from any comment which may appear to be in the nature of carping criticism, for the book has great merit; the descriptions of disease are concise and all the statements, as far as we have been able to test them, are thoroughly reliable. We take pleasure in commending the book to students and general practitioners, for whom it has been especially designed and written.

B. SACHS.

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THE
Journal
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Original Articles.

A CASE OF TACTILE AMNESIA AND MIND
BLINDNESS.

By CHARLES W. BURR, M.D.

Mrs. B. L., sixty years old, colored, was admitted to the Polyclinic Hospital by Dr. Schneideman on August 8th, 1896, complaining of difficulty of vision. The examination of the eyes gave negative results and she was transferred to my care. There is nothing of interest in her family history except that her mother and one brother died of palsy, her father in a fit. The patient has always been a strong, healthy working woman and is the mother of fifteen children. Her present trouble began suddenly about one week before she came to the hospital. While sitting at supper, vision began to fail and decreased so rapidly that at the end of two days she could not distinguish objects at all. At the same time there was numbness of the upper lip and slight frontal headache. Both these symptoms soon passed off.

Examination.—She is a fairly well nourished old woman. Though not at all insane, she is usually dull and apathetic, lying quietly in bed, talking but little and showing interest in nothing. At times, however, she will brighten up and chat with the other patients and visitors. She is perfectly coherent in speech, understands all that is said to her, and has no difficulty in replying. How much of her stupidity is congenital, or as one may say, natural,

and how much due to disease, I am unable to say, but I think the former is by far the larger element, and the latter due to the shock caused by her sudden affliction rather than to any definite brain lesion.

The patient complains only of difficulty of vision, saying simply, "I cannot see." This, however, cannot be her real trouble, since in walking she avoids obstacles, and can tell when an object is placed before her, though she can not recognize what it is, its shape or its color. Dr. Jackson kindly examined her eyes and reports: "Low hypermetropia; pupils react moderately to light; arcus senilis complete; media otherwise clear; retinal vessels small, but discs of good color; fundus normal; apparently no fault with ocular muscles, but patient refuses to fix." Dr. Schneideman found "No apparent disease of eyes to account for poor vision. The parts of the eyes appeared normal and there was no disease of nerve or fundus."

She staggers slightly in walking and is evidently weak, but there is no true palsy. There is slight swaying while standing. The knee-jerks are somewhat below the average in extent of movement and are readily reinforced. The plantar jerks are increased. Voluntary speech is normal. She was never taught to write. Touch, pain and temperature senses are normal in the arms, legs and face, and she localizes sensations correctly, but with the hands fails to perceive objects by touch. With the feet she can distinguish the bare floor from a rug. If a pair of scissors is put in her hand she can not tell what it is, and when asked if it is a knife, says, "Yes, because it feels sharp." She cannot recognize a watch put in her hand, but when it is held to her ear says immediately, "It is a watch, I hear it tick." She calls a match a pencil and makes many similar mistakes. This loss of tactile perception is not, however, absolute. For example, she recognizes a finger when she grasps it, and when I give her my hand on meeting her she shakes it naturally. Again, she always recognizes a silver half-dollar, but one, five, ten and twenty-five cent pieces are all called "quarters." When in bed she grasps the sheet and draws it up, evidently knowing what she has hold of, but she can not feed herself because she does not know what to do with knives, forks and spoons. She can button her clothes, but if handed a loose button does not know what it is. Taste and smell are normal. Hearing is good and, as stated above, there is no motor aphasia. The bladder and rectum

are under complete control. Repeated examination of the urine fails to reveal albumen or sugar. The thoracic and abdominal organs are normal. She was discharged from the hospital September 5th, 1896, in the same condition as when admitted.

Remarks.—There are two points of great interest in this case: Mind blindness, for such I take it is the visual trouble rather than ordinary blindness; and the loss of tactile perception. I wish especially to speak of the latter. It must be very rare, or at least very infrequently observed. For my own part I have seen it only once before. It was a case reported by Dr. Weir Mitchell in the "Medical News" (Vol. 62, p. 101, Oct., 1892). By the courtesy of Dr. Mitchell I had frequent opportunity to examine the patient. She was a married woman, forty-two years old. Her illness began five years before she came under observation with difficulty in seeing large objects, though small things were seen and recognized without trouble. For example, she could see a letter but not an entire word. Glasses gave no relief. During the second year she lost in great degree the power of recognizing colors. She could walk well and avoided obstacles. Dr. Pershing, of Denver, examined her and found she could tell light from darkness. The pupils were equal and each reacted to light thrown in the other eye. The fundus was normal. She walked well and found the knob of the door without feeling for it. In October, 1892, Dr. Mitchell examined her and found the following: The facial expression was vacant. She walked with apparent ease and rarely stumbled. She said she could not tell objects placed before the eyes. She did not recognize a watch by touch, but did by the ear. She did not recognize a clothes brush or pencil by touch. At the first examination she failed to recognize coins, but later did so without difficulty. Touch, pain, temperature and taste senses were normal. She could distinguish the odors of cologne, benzine, alcohol and oil of turpentine, but did not know them by name. She could write her initials and the first part of her name. She could not write isolated letters, except C and O. Spontaneous speech was normal.

Dr. de Schweinitz reported: "Squint in the right eye. The pupils are large and react slowly to light, the right very sluggishly. The discs are grayish red. The veins are full. The arteries small. There is lack of fixation, but the

patient sees light in all directions. The right eye open (left closed) perceives objects to the left of the median line. The left eye open (right closed) perceives objects to the left. There is partial right lateral hemianopsia. She matches blue but confuses red, green and all other colors."

What inability to perceive objects by touch without any affection of the primary tactile sense depends upon, we do not know. It is analogous to mind blindness; so analogous that the discoverer of the latter might have assumed the existence of the former. Just as in mind blindness the patient sees but does not perceive, because he has lost the stored-up results of previous visual stimuli; has lost, for example, the many sensory impulses that go to make up the visual perception of a knife, while the mere raw material of a perception, the sensations, are felt; so our patient has lost the tactile mental images, as we must call them till increased knowledge gives us a better name, stored up from passed tactile experiences, and, therefore, having nothing with which to compare the sum of sensations received, when, for example, she holds a knife, having no memory of what the sensations were when she held a knife before, she cannot perceive it unless some other sense beside touch is brought into use. The condition thus seems to be a memory loss, a partial amnesia. There are other possibilities however. For example, the trouble may be in grouping together the many sensations received from one object by touch, the making of them into one whole, rather than in the loss of old mental images with which the new are in health compared. I think we may assume that some definite area of the brain is concerned with tactile mental images, just as certain areas are concerned with mental hearing and mental vision, but where it is located we are at present entirely unable to say.

SCLEROSIS OF THE CORNU AMMONIS IN EPILEPSY.

By W. L. WORCESTER, M.D.,

Assistant Physician and Pathologist, Danvers Lunatic Hospital.

(Continued from April Number.)

Case 7.—E. R., a negro girl, aged 13, died July 6, 1891, after a residence of two years and five months in the Arkansas asylum, of pulmonary tuberculosis. When received, her right leg was flexed, by contraction of the hamstring muscles, to such an extent as to make walking very difficult, and the contracture increased during her stay until the limb could not be extended much beyond a right angle. She was imbecile to a considerable extent, but could talk very well, was very observant about many things and never gave evidence of any defect of vision or other anomaly of sensibility, general or special. She was stated to have been subject, before admission, to convulsive seizures occurring about once a month, but was only known to have three or four while under treatment.

At the autopsy, there was found to be great atrophy of both occipital lobes of the cerebrum. The supramarginal gyri were small, but apparently healthy; the angular and the occipital convolutions of the external surface of both hemispheres formed an irregularly puckered mass, in which the convolutions could not be distinguished. The same was the case with the cuneus, most of the præcuneus and the occipital part of the occipito-temporal convolutions of the right hemisphere. In the left, the paracentral lobule was much atrophied; the place of the præcuneus was occupied by a cyst containing about two drachms of fluid, and the cuneus reduced to a translucent membrane with some streaks of cerebral matter running over it. The posterior cornua of both lateral ventricles were much dilated, and the cornua ammonis were unduly small—the left distinctly smaller and harder than the right. On median section of the cerebellum, marked sclerosis of the white matter of the arbor vitæ was evident, and the right corpus dentatum cerebelli was also sclerotic.

Microscopical examination of the right hippocampus shows interruption of the stratum pyramidale just before its entrance into the nucleus fasciæ dentatæ. The latter contains pretty numerous characteristic cells, but is irregular in shape, and of not more than half the normal size. The stratum granulosum is almost entirely wanting in some places. In the left, the stratum pyramidale and nucleus fasciæ dentatæ are both almost deficient in characteristic cells, except near their junction, and the nucleus is very small and irregular in shape. The stratum granulosum is pretty well preserved.

In the occipital lobes, the degree of atrophy varies from a condition in which the gray matter seems pretty well preserved, but the medullary portions of the convolutions much shrunken, to a complete absence of nerve-cells and fibres. The atrophied portions are occupied by neuroglia, with but few and inconspicuous spider cells. The histological appearances of the atrophied portions here and in the cornua ammonis are identical.

Case 8.—G. M., white, male, aged 34 at death. Inmate of the Arkansas asylum for ten weeks. The only pertinent information obtained as to his previous history was the statement that he had at some time suffered from "over-heat," and had been subject to convulsions for fourteen years. His mind was inactive, but he was capable of coherent conversation and conformed readily to the regulations of the ward. He had rather frequent attacks of grand mal, controlled to a considerable extent by bromide. There was no paralysis, and no aphasia. He died August 23, 1893, of dysentery.

The brain weighed forty-four ounces. A patch of atrophied and sclerotic tissue extended the whole length of the right first temporal convolution, involving the greater part of the supra-marginal gyrus, and part of the island of Reil. In parts there was a brownish discoloration of the cortex; elsewhere, although shrunken and abnormally hard, it retained its normal color. In the left hemisphere, a similar patch, nearly two inches in the horizontal and one in the vertical direction, was found in the superior and inferior parietal lobules, and another occupying the tip of the temporal lobe and extending over the anterior two-thirds of the first temporal gyrus. The cornu ammonis of that side was also much smaller and firmer than that of the right, which seemed normal.

Microscopically, the atrophied portions of the temporal and parietal regions are made up of a belt of neuroglia fibres, in some parts dense, in others loose, with pretty numerous nuclei, but few large spider cells. Similar tissue occupies the stratum pyramidale, stratum lacunosum and nucleus fasciæ dentatæ of the left cornu ammonis, dense in the nucleus and very loose in the other portions. The nucleus is almost destitute of nerve-cells, and the external half of the stratum granulosum is almost entirely wanting.

Case 9.—R. M., white, female, aged 43 at death. Inmate of the Arkansas asylum for nearly seven years. She was stated to have suffered from puerperal convulsions in 1876 and to have been insane ever since. The date of commencement of her epilepsy was not stated. At the time of her admission she was entirely blind, denying any perception of light. Ophthalmoscopic examination by myself and Dr. T. E. Murrell, failed to disclose any lesion sufficient to account for the blindness. She had rather infrequent but severe convulsions. Mentally, at the time of admission, she was considerably demented, but cleanly in her habits and capable of answering simple questions. Her dementia gradually increased, and in July, 1891, she was noticed to have some paresis of the legs. The knee-jerks were normal or nearly so, and no evidence of anæsthesia could be found. The paralytic trouble increased rapidly; by September 1 she was unable to walk unaided, and by December 10 of the same year she could not stand alone. In walking with support she raised the feet high and set them down flat. In the course of the following year she became entirely helpless, as far as the lower extremities were concerned, and a moderate talipes varus of the right foot developed. She still seemed to have full control of her hands. Mentally, she was almost completely demented, extremely irritable, and disposed to resist everything that was attempted with her. On this account it was difficult to make an ophthalmoscopic examination, but Dr. F. Vinsonhaler succeeded in getting a view of the optic disc, and reported white atrophy of the optic nerve. She died March 14, 1894, of gangrene of the lung.

The brain was evidently considerably atrophied, and there was a large amount of sub-arachnoid fluid. It weighed 32½ ounces. The right occipital lobe presented a large, sunken patch, of brownish color, occupying almost the whole of its external surface, and extend-

ing over its upper border to the internal surface. involving the anterior third of the cuneus, the whole præcuneus, and extending in a narrow strip for about one and a half inches along the sulcus calloso-marginalis. A similar patch occupied nearly the whole surface of the left occipital lobe. The disease extended through the white matter to the posterior cornua of the lateral ventricles. There was a similar, very superficial alteration of the genu of the corpus callosum. The right cornu ammonis seemed harder than normal, but not very much diminished in size; the left was very hard and small. The lateral ventricls were much dilated; their ependym smooth. The optic thalami seemed smaller than normal; the left somewhat flattened.

Microscopically, in the right cornu ammonis, the stratum pyramidale is destitute of nerve-cells, except in a small portion just before its entrance into the nucleus fasciæ dentatæ, which latter, although of nearly normal size, is entirely barren of nerve-cells. The stratum granulosum is well developed. In the region of the lamina medullaris interna is a very loose reticulum of neuroglia. The left cornu ammonis can be described in almost identical terms, except that the nucleus fasciæ dentatæ is much smaller than in the right and contains a very few nerve-cells. Sections of the diseased portion of the left occipital lobe show it to consist of a belt of neuroglia fibres, in some parts dense, in others loose, abundantly vascular, and containing numerous spider cells. Through a considerable extent of its surface a thin layer of cerebral substance seems to have been separated from the subjacent tissue, with which it is only connected by a loose net work, consisting mainly of tortuous blood vessels. The structure of the diseased portion in this region differs from that of the atrophied parts of the hippocampus mainly in the greater abundance of spider cells.

In the foregoing cases it seems to me more reasonable to suppose that the sclerosis of the cornu ammonis is due to the same causes as the other lesions than that it results indirectly from them, as an effect of the convulsions. This, however, is not a matter capable of demonstration, and each one must form his own opinion from the evidence, which I have tried to present fairly.

I have found organic lesions, not accompanied by disease of this convolution, in four other cases of epilepsy. In one, a case of infantile hemiplegia, the left hemisphere,

which was not enlarged, forming a mere thin shell over the enormously enlarged lateral ventricle. In another, there was a cyst in the posterior part of the first left frontal convolution. The third had a small focus of softening in the posterior part of the Island of Reil, and another in the posterior part of the first temporal convolution, both on the left side. The fourth, a case of tumor involving the temporal lobe, will be detailed later.

To discuss the bearing of some of these cases on the theory of cerebral localization is aside from my present purpose.

Whatever may be thought of the relation between the disease of the cornu ammonis and of other parts of the brain in the foregoing cases, I presume it will not be questioned that the anatomical changes, taken together, were, probably in all of them, the cause, rather than the effect, of the convulsions—that they were cases of “organic” as distinguished from “essential” or “idiopathic” epilepsy. All but one of them presented during life symptoms pointing to cerebral disease, other than convulsions. The following eleven cases, on the other hand, showed no symptoms by which they could be distinguished from others in which no gross anatomical change could be discovered. All of them were cases of chronic epilepsy, subject to more or less frequent attacks of *haut mal*. Whether the lesions found are due to the same cause in the two sets of cases may be open to question, but they are, anatomically, to all intents and purposes, the same.

Case 10.—R. H., white, male, aged 26 at death. Said to have been insane four years at the time of his admission to Arkansas asylum, March 11, 1889. No information furnished as to the duration of his epilepsy; the only statement made in regard to it was that he was subject to fits “after drinking whiskey.” Died Sept. 6, 1890, of diarrhœa.

At the autopsy the only lesion discovered was in the right cornu ammonis, which was abnormally small and hard.

Microscopically, the nerve-cells of the stratum pyramidale and nucleus fasciæ dentatæ of this convolution were found to be almost entirely lacking and these portions much reduced in size. The stratum granulosum was well developed. The left hippocampus and other convolutions, so far as examined, presented no important alterations.

Case 11.—P. V., white, male, aged 25 at death; resident of the Arkansas asylum for five years. Duration of epilepsy not stated; insanity said to have shown itself at twelve years of age. He was extremely demented, restless and excitable. Died Aug. 21, 1891, of epilepsy.

At the autopsy, the brain appeared healthy with the exception of the left cornu ammonis, which was sclerotic.

Microscopically, in this convolution, the stratum pyramidale is obliterated for a short distance near its origin, by the union of two bands of sclerotic tissue, starting, one above, the other below the gray matter of the subiculum. During the remainder of its course, it is represented by a small number of irregularly placed cells. The nucleus fasciæ dentatæ is almost entirely deficient in nerve-cells, and the stratum granulosum much thinned. In the other convolutions examined, some spider cells were found in the white matter, and, in portions, some proliferation of neuroglia in the most superficial layer of the gray matter.

Case 12.—C. C., mulatto, male, aged 40 at death; inmate of the Arkansas asylum for five years. Duration of epilepsy unknown. Subject to rather infrequent but severe convulsions. Moderately demented; worked pretty steadily at rough labor. Death occurred Sept. 5, 1891, in status epilepticus.

The only cerebral lesion discovered at autopsy was atrophy of the right cornu ammonis.

Microscopical examination of the affected convolution showed only few and scattered nerve-cells in the stratum pyramidale and nucleus fasciæ dentatæ, which took the carmine stain more deeply than the surrounding tissues. The stratum granulosum was well preserved. In other convolutions examined, slight proliferation of neuroglia was found in the most superficial portions and in the white substance.

Case 13.—M. C., white, male, aged 29 at death. Said to have been subject to epilepsy since four years of age. He was extremely demented and suffered from frequent and severe convulsions, in one of which he died Sept. 12, 1891.

Atrophy of right cornu ammonis was found post-mortem.

Microscopically, the stratum pyramidale is destitute of nerve-cells, except for a short distance just before its entrance into the nucleus fasciæ dentatæ, which is greatly

reduced in size and contains scarcely any nerve-cells. Stratum granulosum is almost destroyed in its inner and lower portions. In the other convolutions the changes were of the same kind as described in the preceding two cases.

Case 14.—B. J., mulatto, male, aged 32 at death. Inmate of the Arkansas asylum for ten months. Said to have become epileptic at eight years of age. Extremely demented and filthy in his habits. Attacks of grand mal moderately frequent. Died March 25, 1893, of pyelonephritis.

There was no appearance of general atrophy of the brain, which weighed forty-four ounces. The right hippocampus major was atrophied in all its dimensions—less than half as broad as left. The type of convolution was somewhat irregular; the interparietal and first temporal fissures were bridged on both sides.

Microscopically, in the right cornu ammonis, the stratum pyramidale and the nucleus fasciæ dentatæ are both almost entirely destitute of nerve-cells, and the stratum granulosum absent in the greater part of its extent. In specimens stained by Pal's method, the deep layer of the alveus and the medullated fibres of the hilum of the fascia dentata are much smaller than usual.

The left hippocampus seems normal, with the exception of an appearance of slight sclerosis for a short distance in the stratum pyramidale.

Case 15.—C. C., white, female, aged 38 at death; resident of the Arkansas asylum for nine years. Duration of epilepsy not stated. She was very much demented, violent, and at times manifested katatonic symptoms, standing for hours together in constrained positions, making peculiar gestures, etc. Died March 15, 1893, of cancer of uterus.

The brain was very small, weighing, with the membranes, only 35 ounces, but seemed of normal color and consistency. Right cornu ammonis much smaller and harder than the left.

Microscopically, in the right hippocampus, the nerve-cells of the stratum pyramidale are absent, except in a small portion just before its entrance into the nucleus fasciæ dentatæ, which is also nearly destitute of nerve-cells. The stratum granulosum is greatly thinned in a large part of its extent. The internal layer of the alveus is much

thinner than normal, but there seems to be no great deficiency of nerve-fibres in the nucleus. The other cornu ammonis and other convolutions examined present no very noteworthy anomaly.

Case 16.—S. C., white, male, aged 42 at death. Duration of epilepsy not stated. Had worked as a printer and, during the time of his residence in the asylum was always a pleasant, inoffensive, moderately demented man. Convulsions of moderate severity, not very frequent. Died Nov. 7, 1893, after having been a patient for seven years, of gangrene of lung.

At the autopsy, the brain, which weighed $40\frac{1}{2}$ ounces, seemed healthy, with the exception of the left cornu ammonis, which was much atrophied, and of almost cartilaginous hardness.

Microscopically, there is almost entire deficiency of nerve-cells in the stratum pyramidale and nucleus fasciæ dentatæ, and the stratum granulosum is atrophied to a great extent. The internal layer of the alveus is almost entirely wanting, but medullated fibres are fairly abundant in the atrophied nucleus. In other convolutions, a thickening of the neuroglia on the surface and some development of spider cells in the white matter are seen.

Case 17.—T. McL., white, male, of no occupation, said to have been epileptic since six years of age. Resident of the Arkansas asylum for seven years. He was subject to rather frequent convulsions; his mental condition was characterized by quiet dementia. Died Feb. 9, 1894, of epileptic exhaustion.

At the autopsy, the brain appeared healthy with the exception of the cornua ammonis, which were both abnormally small and hard throughout. In both hippocampi, microscopical examination shows deficiency of nerve-cells in stratum pyramidale and nucleus fasciæ dentatæ. In the right, a few are found for a short distance before the entrance of the former into the latter. The strata granulosa are well preserved in the greater part of their extent. Elsewhere, nothing distinctly abnormal is noticed except slight superficial thickening of neuroglia in some places.

Case 18.—V. T., white, female, aged 20 at death, resident of asylum two years. Said to have been epileptic from childhood, and to have shown mental derangement when seven or eight years old. Much demented, violent and erotic. Died Dec. 15, 1893, of diarrhœa.

Although she was considerably above average size, the brain weighed only $36\frac{1}{2}$ ounces. The right cornu ammonis was much smaller and harder than the left.

Microscopically, in the right cornu ammonis, the stratum pyramidale is fairly well preserved for a short distance before its entrance into the nucleus fasciæ dentatæ; with that exception, it is destitute of nerve-cells, and but few are to be found in the shrunken nucleus. The stratum pyramidale is much thinned, and so is the inner layer of the alveus. Medullated fibres are not very numerous in the nucleus. In the left cornu ammonis, and in other convolutions examined, little that is abnormal can be found except spider cells in moderate numbers.

Case 19.—E. O., a girl, aged 20, died Sept. 25, 1896, in Danvers Lunatic Hospital, of tuberculosis. She was said to have been epileptic since eight years of age, and was extremely demented. Convulsions were frequent and severe.

At the autopsy, apart from very soft consistency, probably due to post-mortem change, no lesion of the brain was found except sclerosis of the left cornu ammonis, which was much smaller than the right and abnormally hard.

Microscopically, the right cornu ammonis appeared entirely healthy. In the left, the nerve-cells of the stratum pyramidale were almost entirely wanting, and only a few scattering ones remained in the nucleus fasciæ dentatæ. Spider cells, in the atrophied portions, were numerous and large. Stratum granulosum well preserved. In other parts of the cortex nothing noticeably abnormal was found except that the large cells of the third layer seemed rather less numerous than usual.

Case 20.—T. F., male, aged 24, died March 12, 1896, in Danvers Lunatic Hospital, of gangrene of lung. He is said to have had a few fits at the age of fifteen months. They then ceased till he was ten years old; since that time they have been frequent, often several in a day. He was much demented and talked with difficulty.

At the autopsy, the only morbid change noted in the brain was marked shrinking and hardening of both cornua ammonis.

Microscopically, both cornua ammonis presented practically identical lesions. Almost complete atrophy of the stratum pyramidale, a few irregularly disposed cells re-

maining in the usual situation, just before its entrance into the nucleus fasciæ dentatæ, which, on both sides, was almost entirely destitute of nerve-cells. Stratum granulosum thinned in spots; numerous nuclei, but no large spider cells in the atrophied parts. Stained by Nissl's method, the remaining cells in these portions seemed normal. By Pal's method, there was great thinning of the nerve-fibres of the deeper layer of the alveus, and of the fibres radiating into the fascia dentata. The remaining cortex appeared normal.

In addition to the foregoing cases, I met with two in which there was a perceptible difference in the size of the cornua ammonis without any decided difference in consistency, in which microscopical examination showed partial atrophy of the nucleus fasciæ dentatæ, and one, apparently normal to the naked eye, in which there was slight change of the same sort.

In considering the relations of this lesion to epilepsy, the first question is, of course, whether it occurs in other than epileptics, and, if so, how often. So far as my own experience is concerned, I have never found, on the post-mortem table, anything which could be mistaken for the condition I have described in any other than epileptics. Since my attention has been turned to the subject, I have systematically inspected the brains which I have examined with reference to this point. The number so investigated at Little Rock I cannot state precisely, but it was certainly above a hundred. During my service at Danvers I have examined fifty-three brains, or, excluding eight epileptics, forty-five.

I have examined the cornua ammonis of these cases microscopically, with the exception of a few still awaiting examination. In two cases of senile dementia, I found similar changes to those described in a slight degree. In one, a portion of the stratum pyramidale, in the other, a small part of the nucleus fasciæ dentatæ was atrophied. The lesion was not, in either case, enough to make any noticeable difference in the size or consistency of the convolution.

Nerander is the only writer on the subject, so far as I have found, who gives any definite statement of having frequently found induration of the cornu ammonis in other than epileptics, and from the abstract of his paper, it seems probable that the lesions in his cases were other

than those I have described. The facts which I have found in my investigation of this subject may be recapitulated as follows:

1. In nearly 50 per cent. of epileptics examined with reference to this point, I have found one or both of the cornua ammonis smaller and firmer than normal.
2. I have failed to find this lesion in any other cases.
3. In a large proportion of these cases, this condition was associated with other abnormalities of the cerebrum, most frequently microgyria of an entire hemisphere.
4. Whether occurring alone, or associated with other morbid conditions, the histological changes were practically identical in all my cases, consisting in destruction of the neurons having their origin in the stratum pyramidale and nucleus fasciæ dentatæ.

Whatever may be thought of the relation between the lesion and the disease with which it is associated, there seem to me singular and interesting facts. Do they warrant any conclusion as to a relation of cause and effect? In view of the frequency of this condition in epileptics, and what I must consider its great rarity in those not subject to this disease, it seems to me that the question of mere coincidence hardly requires discussion. The dilemma is practically presented, whether the epilepsy causes the anatomical changes or they the epilepsy.

As has been seen, the weight of authority is in favor of the former opinion. At the same time, the principle reason for this appears to be rather the improbability from anything we know of the functions of this convolution, that it can have any special relation to epilepsy, than any definite theory as to the way in which epilepsy could bring about such changes in a single convolution. The only attempt at an explanation that I have seen, that of Wandt, already quoted, assumes an entirely hypothetical increase of intraventricular pressure, and offers no explanation of why it should be, in most cases unilateral, or how it could bring about such a condition if it existed. So far as I am aware, no such result has been reported in cases of hydrocephalus, in which intra-ventricular pressure is known to be excessive.

There remains the supposition that the condition of the cornu ammonis is the cause of the convulsions, which does not, to my mind, present any difficulty. We have to do, practically, with a cicatrix involving the whole cortex

of a pretty large convolution. Two questions arise in this connection: (1) Can a cicatrix of the cerebral cortex cause convulsions? and, (2) Is this liability limited in any such way as to exclude the convolution in question? That irritations of the cortex may cause convulsions is not, I suppose, questioned at the present day. There is abundant evidence, both clinical and experimental, that such may be the case. That a cicatrix of the cortex may act as a focus of irritation is also, I suppose, pretty well made out. I myself have had a case of Jacksonian epilepsy in which the lesion found was an atrophied state of the cortex in the upper part of the fissure of Rolando, histologically very similar to that found in these cases. There remains, then, the question whether an irritation of this particular convolution can be the cause of convulsion. It is not probable that cases often occur in which lesions, other than the one under discussion, are strictly confined to the cornu ammonis. I have met with but one of this description in my reading. (Meynert's 17th case).

"Brain of an epileptic; personal data wanting. Cysticercus of choroid plexus of left cornu ammonis, which is, as it were, soldered to the alveus of the cornu ammonis in an extent of more than a centimetre, which spot appears infiltrated and surrounded by a thick injection of blood-vessels."

Cases are not lacking, however, in which convulsions have been caused by lesions of the temporal lobe.

In case No. 74 of Sommer's collection, it is stated that epileptic attacks and insanity followed an acute cerebral disease. Convulsions usually nocturnal. A medullary cancer, originating in the anterior portion of the right cornu ammonis, has invaded the posterior and middle lobes of the cerebrum.

Bowerman reports¹² a case in which a fracture of the skull from a blow behind the right ear, at the age of fourteen, was followed by chronic epilepsy, beginning four years after the lesion. Post-mortem, a depression of temporal bone was found, with a corresponding depressed cicatrix about two inches in diameter, involving the superior and middle temporo-sphenoidal convolutions.

I myself saw a case, at the Arkansas asylum, of what appeared to be epilepsy, lasting about four years, without

¹²N. Y. State Hospitals Bulletin, Vol. I., p. 385.

any other symptoms except some headache calculated to excite suspicion of any organic lesion, in which, at the autopsy, a psammoma was found, about two inches in horizontal by one-half in vertical diameter, growing from the dura at the tip of the left temporal lobe, which was softened in the greater part of its extent.

There seems, then, to be positive evidence, if it is needed, that irritation of the temporal lobe may excite convulsions. Such being the case, it seems to me most reasonable to conclude that the scar in the cornu ammonis is, in these cases, a focus of irritation which gives rise to the convulsions. I do not suppose that this convolution has any special prerogative in this respect. It seems to me that we are warranted in believing that a scar in any part of the cortex may have such an effect. Certainly, for my own part, if I should find, in a case of epilepsy, a lesion of the same character and extent in any convolution I should have no hesitation in accepting it as a sufficient cause for the convulsions. In my cases in which this convolution has been associated with others in morbid processes, I do not attribute to it any more influence than to the others in causing convulsions. But where, as in a number of my cases, no other lesion can be discovered, it seems to me entirely justifiable to accept the changes in the cornu ammonis as the starting point of the epilepsy.

Whether as cause or effect of the symptoms with which it is associated, it is evident that this convolution is peculiarly subject for some reason to a very definite disease. If the pathology of this condition could be determined, it would, doubtless, go far to settle the question of its relation with unilateral atrophy or arrested development of the brain, in cases of infantile cerebral palsy. If it is, in such cases, to be considered only a part of the general disease of the hemisphere, there would seem to be nothing improbable in supposing that the same cause, whatever it may be, which in one case affects the entire half of the brain, may, in another, act only on one or a few convolutions. The symptoms would, of course, vary with the extent and situation of the disease, and if it were situated outside of the motor area, it might betray itself only by convulsions. In view of the identity of the lesion of the cornu ammonis in my cases of unilateral cerebral atrophy with those in which the disease was confined to the cornu, I cannot help strongly suspecting that there may be a like similarity in the causes.

Unfortunately, the etiology of unilateral atrophy is obscure. Of twelve cases collected by Richardiere,¹³ the disease began in seven with convulsions followed by hemiplegia. In five, paralysis occurred, either congenitally or in infancy, and there is no history of convulsions at the time. For the convulsions themselves, no cause is assigned, with the exception of one case, in which they are said to have followed measles.

Neuroth¹⁴ reports five cases of infantile cerebral paralysis following whooping cough, which he believes to be due to hæmorrhagic encephalitis. In view of the fact that such paralysis often follows infectious diseases, Strumpell's hypothesis of a polio-encephalitis seems as probable as any, but it must await confirmation by autopsies on recent cases. The probability is that children not infrequently die either during or soon after the onset of paralysis, and it is very desirable that such cases should be thoroughly examined. Of this, naturally, there is no opportunity in a hospital for the insane.

Supposing it to be the fact that sclerosis of the cornu ammonis may be a cause of epilepsy, a practical conclusion may be drawn from it. A large proportion of what appear, clinically, to be "essential" epilepsies, are upon this supposition of organic origin. This would be sufficient to account for their incurability, and to render it improbable that any drug will ever be discovered which will permanently cure the symptoms of a lesion in its nature irremediable.

Whatever may be thought of the correctness of the conclusions to which I have been provisionally led, the frequency of the lesion, in connection with epilepsy, and the uniformity and definiteness of its localization, are, it seems to me, matters of great interest, and worthy of further study with a view to the elucidation of the relations, whatever they may be, between it and the associated symptoms.

In conclusion, I wish to make it clear that I do not believe that this convolution has any more to do with epilepsy than any other, apart from the evident fact that it is peculiarly liable to disease. Neither would I be understood as asserting that all epilepsies originate in any part

¹³Etude sur les Scleroses Encephaliques Primitives de l'Enfance, Paris, 1885.

¹⁴Wiener Med. Wochenschrift, 1896, No. 16.

of the cerebral cortex. It seems certain that epileptiform convulsions may be due to peripheral irritations, and to toxæmic conditions, and epilepsy, as we know it, consists in the repetition of such convulsions. I think it altogether probable that many epilepsies depend on such causes. But it does not seem to me necessary to assume, without evidence, such a cause, or to cover our ignorance by saying that epilepsy is a functional disease, in cases in which there is an entirely different anatomical explanation for the symptoms.

DESCRIPTION OF THE DRAWINGS.

Fig. I.—Left cornu ammonis of case 14. Carmine stain. Apparently normal, except for a narrow band of sclerosed tissue, extending from x to y.

(fi) fimbria.

(stpy) stratum pyramidale.

(nfd) nucleus fasciæ dentatæ.

(stgr) stratum granulosum.

Fig. II.—Right cornu ammonis of same case. Sclerosis of stratum pyramidale and nucleus fasciæ dentatæ, with atrophy of their nerve-cells and of the stratum granulosum. References as above.

Fig. III.—Left cornu ammonis, same case. Pal's stain. Normal, or nearly so.

(nfd) nucleus fasciæ dentatæ.

(hfd) hilum fasciæ dentatæ.

(alv) superficial layer of alveus.

(a) deep layer of alveus.

Fig. IV.—Right cornu ammonis, same case. Absence of medullated fibres of hilum fasciæ dentatæ and deeper layer of alveus. References as above.

The drawings for the first three figures are by Dr. Ira Van Gieson, to whom I desire to return my hearty thanks.

ADDENDUM.

Since this article was in print I have found, in both cornua ammonis of a patient dying, in the Danvers Lunatic Hospital, of general paresis, changes identical with those above described. There was no history of epilepsy and the patient did not suffer from convulsions during his stay in the hospital. This would seem, therefore, to be an instance of the occurrence of this lesion independently of convulsions. Judging from the microscopical appearances, I incline to think it of older date than the general paresis.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, Feb. 2, 1897. B. Sachs, M.D., President.

CASE SIMULATING SYRINGOMYELIA.

Dr. Graeme M. Hammond presented a lady who, ten years ago, after an illness similar to an attack of la grippe, began to have loss of sensation to pain and temperature in left arm. During these ten years the symptoms had extended to the left leg, trunk and side of the face. Dr. Hun had carefully examined her, and had made a diagnosis of syringomyelia. When first seen by the speaker the symptoms had been certainly very like those of that disease—absolute insensibility to temperature and pain, with perfect preservation of the sense of touch. But the absence of paralysis and contractures, the normal electrical contractility and the fact that the other side was not affected argued against this diagnosis. In addition to this, there were several symptoms of neurasthenia—insomnia, noises in the ears and confusion of ideas. It had occurred to him that the case might be of neurasthenic origin, and acting upon this theory, she had been given bromides, together with as much mental impression as possible. The result had not been disappointing. Now, the pain and temperature sense had returned in the face and trunk. The sensation of pain and temperature were not fully restored in the left hand or in the trunk, but were decidedly improved. She had never been told what the symptoms of the disease were, so that there could have been no suggestion.

Dr. M. Allen Starr suggested that there might possibly be a small area in the cord of disease that had started the symptoms and given the "suggestion," thus making the case a complex one.

CEREBELLAR ATAXIA.

Dr. George W. Jacoby presented a little girl of nine years—a typical case of cerebellar ataxia. In this case there was no heredity. The father married his own niece, but with this exception the family history was negative. This child did not walk until three years of age, and was generally backward,

both mentally and physically. Examination showed marked ataxia in both upper and lower extremities. When unobserved, the child exhibited constant choreiform movements of the head and upper extremity. When walking the body was thrown forward and the head somewhat backward; the legs were spread far apart, and there was a constant tendency to falling. In reaching for an object, there was an uncertainty of movement. The eyes showed a slight fixation nystagmus at times. There were no changes in the optic nerve. The reflexes were very much exaggerated. There was no deformity in the lower extremities. He felt that the case should be looked upon as a congenital defect of the cerebellum.

Dr. Joseph Collins endorsed the last statement of Dr. Jacoby. He had looked into the literature of the subject, and had been impressed with the necessity of differentiating between hereditary and familiar changes. In a case that he had had there had been no hereditary history whatever, nevertheless a younger child, who had died about the end of the second year, and was described as having had from its birth "St. Vitus' dance" and other symptoms, had probably suffered from the same disease.

DEMONIACAL POSSESSION.

Prof. Wm. James, of Harvard University, delivered an address on this subject. He said that our knowledge of altered personality had made rapid strides in recent years. We had the transient altered personality of epileptic insanity, and certain dream states that had been described under the name of "ambulatory automatism"—the subject going from home and returning after an interval of, perhaps, weeks, with the memory of what had happened during his wanderings utterly effaced. In one case that he had treated, hypnotic suggestion had brought back the memory of the wanderings. There was still another altered personality, that called "spirit control." This was connected with demoniacal possession. The obsolescence of public belief in the possession by demons was a very strange thing in Christian lands, when one considered that the sacred books of our religion were full of this belief. Every land and every age had exhibited the facts on which this belief was founded. The particular form of supernatural origin varied with the traditions and popular beliefs of each country. When the Pagan gods became demons, after the triumph of Christianity in Europe, all possession was looked upon as diabolic. It was now replaced by the thoroughly optimistic belief that changed personality is the spirit of a human being coming to bring messages of comfort from the sunny land. The unconsciousness, the speaker said, was usually ushered in by a more or

less pronounced convulsion—the person's character became entirely changed in its attitude, voice and manifestations. After an hour or two, the manifestation passed off, leaving a complete amnesia behind of everything that had occurred. During the intervals of the attacks the person was entirely well. The condition was, therefore, entirely distinct from any form of insane delusion. Mr. Percival Lowell had reported that in Tokio, Japan, there were a number of persons who cultivated the power of passing into trances. In China there was a widespread belief that possession by gods and spirits could take place. Mr. Nevins, a missionary in China, had reported a number of cases of demoniacal possession. In Japan there was a curious superstition that the person afflicted was not affected by a demon but by a fox. In India instances of this kind were extremely common.

The speaker said that the witchcraft delusion had been explained in various ways, but to him witches were not neuro-pathic persons, but the accusers were. He had carefully examined the witchcraft trials, and had found that it started in some demon disease in the neighborhood. These "demon diseases" were very common in those days, being any functional neuro-pathic disease. If there were no obvious physical disorder, and the symptoms did not yield readily to the usual medical treatment, the case was considered to be one of demon disease. Professor James quoted from a book written in 1602, by a French magistrate, in which a detailed description was given of a girl possessed with five demons, and the manner in which they came out of her mouth and ran about the fire two or three times before disappearing. He said that these descriptions reminded one of the classical hysterical attack—the lump in the throat, the convulsive seizures, etc. The cases appeared to be examples of imitative hysteria, patterned after the case existing at that time. Differences in the different countries, of course, came from the differences in the psychological climate. Many interesting reports had been published of late years of epidemics of chorea, supposed to have resulted from imitation. These epidemics had been known to last for months or even years. An interesting case of demoniacal possession in France, in 1863, had been reported by Dr. Augustin Constance, in Savoy. A similar epidemic had been reported in Italy. The epidemic in France began with hysteria among certain children, and was propagated by example until at last a very large number of persons was attacked with all the symptoms of demoniacal possession. When Dr. Constance arrived upon the scene, a year after the breaking out of the epidemic, 110 persons were affected. He examined a number of these individuals, and found them to be suffering from hysterical attacks, brought on by suggestion. The patients were wisely sent away to other villages, and in that way he broke up the epidemic.

Hystero-demonopathy is the name given to these symptoms. No one could fail to recognize in these attacks the analogy to the performances of the numerous spiritualistic mediums of the present time. It would be strange, indeed, if a phenomenon which had played such a large part in history should have died out without leaving anything in its place. Medical men should learn from all this a certain lesson, *i. e.*, that as our views had become optimistic, instead of pessimistic, the whole thing had become harmless. We live in a day when there is much alarmist writing in psychopathy about degeneration, and the alarming significance of all sorts of symptoms and signs, so that there is danger of drawing the line of health too narrowly.

Dr. C. A. Herter said that the idea of connecting the powers of modern spiritualistic mediums with the peculiar forms of demoniacal possession which occurred in former years, was a most interesting one. This fact had been brought out most interestingly and impressively in the address. He had been much interested in the gradual change from the damaging character to the comparatively beneficent character of these phenomena.

Mr. Martin said that it seemed to him rather remarkable that the suggestions which occurred to the possessed person related almost entirely to ethical matters, or religious subjects. A large portion of the recorded cases that he had met with referred to the possession by devils who were leading the person astray, or into immorality. He would like to ask if Professor James had observed the same thing.

Professor James replied that it was a law of the secondary consciousness that it took the religious form. He had no explanation to offer, however, of this law. It was a singular fact that involuntary writing was apt to take the spiritualistic form. This would occur in the case of persons who had no intellectual hospitality for that view, and who had not been exposed to spiritualistic influences. Spirits, religious truths and philosophical discourses were the staple of these communications.

Dr. Mary Putnam Jacobi said that as in so many cases of melancholia the grief was about having sinned against the Holy Ghost, even in persons who had had no religious or Calvinistic instruction, she would like to ask if Professor James considered it an example of the phenomena just spoken of. She would also like to ask his opinion of an essay that had been published, entitled, "Were the Salem witches entirely guiltless?" According to this essay, although these witches were not possessed by devils, they were abandoning themselves to impulses coming from the lower structures of their natures—the result of ancestral influences.

Professor James replied that he did not think the delusions of melancholia had anything to do with the subject under dis-

cussion. The sin against the Holy Ghost was only an endeavor to explain the grief which was left. Regarding the essay by Professor Barrett Wendell, to which allusion had been made, he would say that at the time of the witchcraft belief there were certainly persons attempting to do what they could by diabolical aid, but in all probability they formed a very small part of it. In Salem, the girls from whom the accusations emanated had been having hypnotic seances from a West Indian slave, who was herself practically insane. They passed then into such a condition that they were accused of witchcraft, and were tried under such circumstances as to impress them powerfully by suggestion. From what we know of imitative hysteria, the whole matter was entirely explicable on that basis, without any supposition of guilt upon the part of these children. It was a suggestive epidemic of a semi-hysterical nervous disorder.

Dr. C. L. Dana said that the speaker had made quite clear the relation of trance to demoniacal possession of old, but he would like to know how widespread was this condition now. He knew that about fifteen years ago spiritualism had been immensely prevalent in the Eastern states. If the condition had continued to exist and spread, there was certainly much more in the United States to-day than in civilized countries several hundred years ago.

Professor James replied that it would be difficult to answer this question statistically, as we had no trustworthy statistics. We knew, however, that at the present time there were many "faith healers."

UEBER FAMILIÄRE SPASTISCHE SPINALPARALYSE. [Concerning the Family Form of Spastic Spinal Paralysis.] Deutsche Zeitschrift für Nervenheilkunde, Band IX., Heft 3 u. 4. By H. Hochhaus.

Three cases of spastic spinal paralysis, occurring in one family, are reported. One child of seventeen years in this family was sound. The disease began in the second year of life, and by the sixth year had acquired great intensity. After this age the symptoms remained stationary in one case, improved in another, and increased in a third. In all three cases spastic paresis of the lower extremities, with exaggeration of the reflexes and foot clonus, without disturbance of sensation or of the vesical and rectal functions, was observed. In one case distinct atrophy of the legs was noticed. No cause could be discovered; birth had not been premature or difficult, nor were there signs of early meningitis. Tuberculosis was hereditary in the family of the mother. The limitation of the spastic paresis to the lower limbs during many years indicated some disease of the pyramidal tracts in their lower portion, developing in imperfectly formed fibres.

In none of these cases were any symptoms observed which must of necessity be attributed to a cerebral lesion. SPILLER.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Nov. 23, 1896. Vice-President Dr. F. X. Dercum in the chair.

Dr. C. W. Burr read a paper entitled

A CASE OF TACTILE AMNESIA AND MIND BLINDNESS.

(See this journal, page 259).

DISCUSSION.

Dr. Wharton Sinkler.—This report of Dr. Burr recalls to my mind a somewhat analogous case which I saw some years ago, and which is still under observation. The patient has distinct hemianopsia, and there is also loss of memory for the names of objects, and to a certain extent there is also loss of tactile memory. If she were given a pencil and asked to name it, she would say that she knew what it was, but could not recall its name. If asked if it were a pencil, she would say, "Yes;" on being given a spoon she would examine it by sight and touch, but was unable to state what it was. If asked if it were not a bottle, she would promptly say, "Yes."

Dr. Wm. G. Spiller.—The condition which Dr. Burr speaks of as tactile amnesia has been described under the name of tactile aphasia. It is similar to the condition known as optic aphasia, which was first described, I believe, by Freund. Dr. Burr stated that when a watch was placed in his patient's hand she would look at it, but was unable to name it until it was placed near her ear. It would seem as though the sensory impulse had to reach the speech area of the cerebral cortex by some tract other than the tactile. In optic aphasia the patient is unable to name an object merely after looking at it. It has been claimed that optic aphasia does not exist alone. At least one of the cases reported by Oppenheim shows that it may be the only symptom of aphasia. These rare forms of aphasia are intensely interesting. So far as I know, there have been but three autopsies in cases of "pure" word blindness. They are those of Dejernie, Wyllie, and Redlich, and it would seem that in this form of aphasia the lesion involves the cuneus, lingual and fusiform lobules. I have in my collection preparations from Redlich's case. I am not aware of any fully satisfactory autopsy in any case of "pure" word deafness. This form of aphasia was first described by Sérieux.

Dr. Mills's case of word deafness was most interesting and of very great importance, but it was not, I believe, a case of "pure" word deafness. There was a lesion in each first temporal gyrus, but I am unable to state at the present moment all the symptoms observed. From the location of the lesion within the cortical speech area of the left hemisphere—judging from what we know of the location of the lesion in "pure" word blindness outside of this area—I would expect to hear of some form of paraphasia or paraphagia. By "pure" word deafness I mean the condition in which there is an inability to comprehend spoken words with no other disturbance of speech. A patient who has this form of aphasia should be able to write, read and talk in a normal way.

Dr. Charles S. Potts presented a case of Primary Neurotic Atrophy.

DISCUSSION.

Dr. F. X. Dercum.—This, I believe, is the second time that this disease has been reported in this country. Two other cases, brothers, were reported by Dr. Sachs of New York, some years ago. This would make the present case the third upon record. I would like in this connection to make a verbal report of a fourth case recently observed by myself in the clinic at the Orthopædic Hospital.

The man visited the institution but once and did not return. The condition was, however, unmistakable. He was some thirty years of age and presented the symptoms of primary neurotic atrophy in a very marked degree. Not only was there present the characteristic peroneal atrophy, but there was also present some atrophy of the muscles of the forearms, and especially of the muscles of the hands. According to the patient's statement, he had some six or eight years before had a sharp pain in one heel, and after this pain had persisted for a time, he noticed weakness in attempting to extend or abduct the foot. At the time that I saw him the weakness of the legs and the foot-drop were so marked that the gait resembled that of a multiple neuritis. There was also some wrist-drop so that the resemblance to a multiple peripheral nerve-palsy was exceedingly suggestive. There were no disturbances of sensation save the occasional sharp pain referred to. No paræsthesias appeared to be present. The progressive and gradual character of the disease, the beginning of the atrophy in the extensor and abductor muscles of the leg, as well as the slowly progressive character of the affection leave no room for doubt. Unfortunately, the patient did not return to the hospital, and the study of the case could not be satisfactorily completed.

Dr. Wm. G. Spiller presented a porencephalic brain from a boy who had been hemiplegic. (See paper on Infantile Hemiplegia published in this journal Jan., 1897.)

Dr. Samuel Wolfe presented a paper on

TETANUS NEONATORUM.

After a difficult labor of considerably more than 24 hours, Mrs. M., aged 22, was on November 5 delivered of her first child. The child is the subject of the present paper, but as the circumstances connected with the pregnancy and delivery of the mother have an important bearing in the case, I will briefly relate a few incidents pertaining to them. The woman's mother is a confirmed hypochondriac. Her husband is a very emotional man, of unusual instability, which is illustrated by the house and remained away for some hours. When he returned fact that while the labor was in progress he ran out of the he created a tumult by his loud weeping and loss of self-control.

During the last month of the pregnancy the mother had considerable myalgia about the chest, and was dominated by a conviction that she had a diseased heart. For this there was no ground, and she was positively assured to that effect.

When the labor came on the pains continued regularly for about ten hours, bringing about sufficient dilatation of the os uteri and bulging of the membranes to warrant a rupture of the latter. From this point there was no further progress. All genuine contractions ceased, though she declared herself as being continuously in agony, and insisted that she would not recover. Quinine, in six-grain doses every two hours, was now given as an oxytocic, until 24 grains had been used. The urine, which was retained, was drawn off by the catheter. An attempt to apply the forceps failed. She was now left for twelve hours in the hope that the contractions would recur, but at the end of that time, the inertia still persisting, the forceps were applied with difficulty with the aid of a consultant, and at the end of a few hours a male child of about seven pounds was delivered. Respiration was only fully established after an hour of faithful manipulation. The forceps had produced a severe abrasion over the angle of the right lower jaw, and a slighter one in the left parietal region.

Next morning it was observed that there was considerable spasm of a tonic character in the muscles of the face, the left eye being spasmodically closed, and the muscles of the lower part of the right side of the face somewhat contracted. On the third day the latter group of muscles was paralytic, the

spasm of the orbicularis palpestrarum had largely disappeared, and the nurse reported that she had occasionally observed spasmodic movements of the left arm and right leg. On the fourth day nearly all indications of spasm and paralysis in the muscles of the face had passed away; the spasms of the leg and arm still occurred, with occasional opisthotonos and spasmodic respiratory movements. On the fifth day it was found that both arms were bent at the elbows, the wrists flexed, the fingers and thumbs clinched; on the right side the thumb into the palm, on the left over the index and middle finger. The legs were extended and the thighs slightly flexed and giving a picture of moderate emprostotonos. This condition continued until death occurred when the child had reached the twelfth day of its life.

Throughout the child had taken but little nourishment. It nursed from the breast, but very indifferently, and all attempts at artificial feeding were only partially successful.

The cord had not separated until the day before the child's death. The forceps abrasion had become covered by the second day with a thick, black incrustation, which before death had fallen off leaving a healed surface.

The medication consisted of the administration of $1\frac{1}{2}$ gr. doses of sodium bromide every two hours, throughout the illness.

The case presented, in the marked involvement of the upper extremities and the absence of trismus, rather the symptoms of tetany than those of tetanus. While tetanus is comparatively frequent in the first weeks of life, tetany is not. The writer will appreciate a free discussion of the diagnosis. The possibility of a closer relation than is ordinarily conceded between these diseases might be considered. The fact that tetany usually follows exhausted conditions from prolonged irritation and disease of mucous membranes, and tetanus is associated with traumatism, in which the skin and subcutaneous tissues are involved, might lead to theories as to susceptibility of various sensory tracts.

Dr. J. M. Taylor made some remarks on an epidemic of poliomyelitis occurring last August in Maine. The cases were seven in number, so far as collected, and all occurred at the same place and within one week. One case was fatal. The subject will be presented more fully at the meeting of the American Neurological Society in May next.

Dr. Wharton Sinkler.—There have been several epidemics of poliomyelitis reported, but in none has any source of infection been found. The fact that there have been epidemics of this disease strengthens the belief that in most cases of poliomyelitis there is an infectious origin. Some years ago I called attention to the fact that poliomyelitis occurs four out of five

times in the summer months. There may be something connected with the heat, or with putrefactive changes favoring the development of an infecting agent, which finds its way into the system.

Dr. Wm. G. Spiller.—I have had the opportunity of studying the changes which occur in the spinal cord in this disease. The case was one reported by Redlich. The child lived about ten days after the beginning of the process. The small spinal vessels were very full of blood, and numerous capillary hemorrhages were found. The anterior horns were especially affected. The ganglion cells seemed to be altered secondarily. The findings confirm the theory of an infectious origin, which Dr. Sinkler has just referred to.

Adjourned.

ZWEI FAELLE VON ASTHENISCHER BULBAERPARALYSE. [Two Cases of Asthenic Bulbar Paralysis.] Deutsche Zeitschrift für Nervenheilkunde, Band IX., Heft 3 u. 4. By A. Kojewnikoff.

Kojewnikoff describes two cases of this rare form of bulbar paralysis; the first he regards as typical, the second, however, presented some unusual features. Alcohol seems to have been a causal factor in the first case. A diminution of vision, manifested especially by a rapidly developing decrease in the size of the visual fields on examination, due to exhaustion of the retina, and a similar condition of rapid exhaustion in the sense of taste were observed in the second case.

It is possible that these phenomena may have been due to hysteria, but no other changes of sensation were noticed. The ciliary muscle and the iris presented the phenomena of exhaustion. Another unusual feature was the reaction of degeneration in the muscles of the tongue and soft palate. Sugar was also observed in the urine. This was supposed to be due to a lesion of the diabetic centre in the oblongata.

[Attention may be called to a case of this disease occurring in America, and reported by Joseph Collins in the April number, 1896, of the International Medical Magazine.]

SPILLER.

VOLITION. From Mind, July, 1896. By G. F. Stout (Editor, Mind).

Stout holds that the triumph of the voluntary impulse over conflicting motives is not like the stronger over the weaker of two opposing forces applied to a particle which moves in the direction of the stronger force, yet is retarded by the weaker one. In the mind act, he insists, the conflicting motives are driven off the field, and simply become, if anything, external obstacles in the way of attainment, that is, they are outside the sphere of deliberation, or, in other words, things that were motives, cease to be motives because they are no longer desires. He defines an involuntary act as "one which takes place in opposition to a voluntary resolution which exists simultaneously with it and is not displaced by it."

CHRISTISON.

Periscope.

With the Assistance of the Following Collaborators:

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ANATOMY AND PHYSIOLOGY.

SUR LES FIBRES DE PROJECTION ET D'ASSOCIATION DES HÉMISPÈRES CÉRÉBRAUX (Concerning Projection and Association Fibres of the Cerebral Hemispheres). *Comptes Rendus Hebdomadaires des Séances de la Société de Biologie*, February 20th, 1897. By J. Dejerine.

Dejerine refers to the report made by him in 1893 to the Society of Biology, in which he stated that his investigations of twenty-three cerebral hemispheres with cortical lesions demonstrated that the anterior three-fourths of the frontal lobe and the occipital lobe, including the angular gyrus, send no fibres to the crusta, but that all the crustal fibres come from the middle portion of the cerebral cortex (Rolandic region, paracentral lobule, foot of the three frontal convolutions, anterior part of the parietal lobe, middle portion of the temporal lobe) without interruption in the central ganglia, and without receiving additional fibres from these. In the same paper he stated that the internal bundle of the crusta arises in the Rolandic operculum and the foot of the third frontal convolution, and that the external bundle arises in the mid-portion of the temporal cortex, especially in the second and third convolutions.

In his present report he states that in an examination of three cases of superficial cortical lesions in the middle and anterior regions of the frontal lobe he was able to observe distinct degeneration of the anterior limb of the internal capsule and atrophy of the internal thalamic nucleus. In another case in which there was a superficial lesion of the angular gyrus, and the sagittal layers of fibres were not involved in the primary lesion, he was able to trace the degeneration into the pulvinar and the external thalamic nucleus. Secondary degeneration shows also that the lingual and fusiform lobules give origin to projection fibres which pass to the posterior and inferior part of the thalamus.

The views of Flechsig concerning the association centres are not, therefore, admissible. SPILLER.

OBSERVATIONS ON THE HISTOLOGICAL DEVELOPMENT OF THE CEREBELLAR CORTEX IN RELATION TO THE FACULTY OF LOCOMOTION. Aurelio Lui. *Revista Sperimentali di I. Freniatria* Vol. XXII., fac. 1.

The article is a continuation of work already published by the author and records a series of observations made before and after

birth upon the cerebellum of certain birds and animals. The rapid Golgi method was used, and the subjects were sparrows, starlings, rabbits, cats and dogs.

The object of the work was to find out whether or not there was any correlation between the development of the power to walk and the histological structure of the brain, and if so to discover the nature of the changes that took place in the nerve centres while the function of walking was being acquired.

According to the author's observations, the power to walk, as gradually acquired, is accompanied by a progressive modification of the Purkinje cells from the pyramidal type of the young to the more fully rounded type of the adult. The external granular layer is also gradually reduced in thickness, thus leading to the conclusion that there is some such correlation as suggested.

The histology of the cerebellum is treated in a number of details bearing upon the subject. The conclusions which the author reaches may be summed up as follows:

(1) The whole of the external granular layer cannot be regarded as an embryonic stage of the molecular substance.

(2) Development takes place in the internal granular layer independent of the animal's increasing ability to stand or walk.

(3) As the locomotor functions are acquired the Purkinje cells and the cells of the deep layers of the granular zone show the most marked and regular development. It is, therefore, concluded that the Purkinje cells are connected with the power of locomotion.

(4) The correlations between anatomy and function which are the most clearly established are those existing between the processes of Purkinje cells and the ramifying plexuses on the one hand and the descending dendrites of the basket cells and the bodies of the Purkinje cells on the other.

JELLIFFE.

THE EFFECTS OF THE LOSS OF SLEEP. By Professor G. T. W. Patrick and Dr. J. Allen Gilbert (Studies from the Psychological Laboratory of the University of Iowa).

These experiments are supposed to be the first of their kind on human subjects. In 1894, at the International Medical Congress, held at Rome, M. de Manacine reported his experiments on young dogs he kept from sleeping and which died from insomnia at the end of the fourth or fifth day. These University of Iowa experiments were conducted upon three instructors under thirty years of age, of good health and habits, who were kept awake continuously for about ninety hours. They were constantly attended by one or two watchers. They took their regular meals at 7 A. M., 12.30 P. M. and 6 P. M., also a light lunch at 12.30 A. M. Their food was normal in character and amount and their time was mostly engaged in reading, games (both light and active), walking, working upon apparatus, and about one-third of the whole time was occupied with the experiment observations, which were taken every six hours and took two hours each time.

Case 1. An assistant professor, aged twenty-eight, single, health perfect, nervous temperament, of great vitality and activity accustomed to eight hours sleep. By the second night he did not feel very well and was very sleepy. The third night he suffered less, but had a hallucination of sight in the form of a greasy-looking molecular layer of rapidly moving or oscillating particles appearing on or near the floor. On the fourth day and evening he felt well. The daily rhythm showed the sleepy periods to be from midnight to noon, but most marked about dawn. The visual hallucination after the second night had gradually changed from being a layer of particles to a swarm of little bodies like gnats, but colored red, purple or black, and did not change with the position of the eye. Meanwhile his vision

was growing acuter until he went to sleep, after which the hallucination had vanished. The subject retired after ninety hours waking and immediately fell asleep, but was hourly roused (to test the depth of his sleep) and each time immediately lapsed into profound sleep. After ten and a half hours sleep he awakened spontaneously, felt wholly refreshed and as well as ever. The following night he slept two hours longer than usual. At the conclusion of the sleep fast the subject had gained twenty-seven ounces in weight, but after his ten and a half hours sleep he had lost thirty-eight ounces in weight. Although acuteness of vision gradually increased up to the time of sleeping, it fell below the normal after sleep. Voluntary motor ability decreased (tested by rapid tapping of the forefinger on a key connected with a recording drum and graphic chronometer). The pulse, which was eighty-eight the first evening, gradually lost until it was sixty-one, the fourth evening, but the pulse, which after fatigue the first evening was eighty-nine, dropped to fifty-two after fatigue on the fourth evening. Sensibility to pain in both upper and lower thresholds, gradually decreased slightly, but returned to the normal on the fourth evening a few hours before sleeping. Memory gradually declined but returned to the normal the last evening, and after sleep it dropped more than two-thirds. Attention gradually waned (as tested by the adding of figures) but was approaching the normal on the fourth evening and after sleep it was fully restored. Grip and pulse slightly decreased. Reaction time somewhat increased and returned to the normal after sleep. His temperature was hardly changed.

Case 2. An instructor, aged twenty-seven, single, quiet, of excellent health and accustomed to nine hours sleep.

Case 3. An instructor, aged twenty-four, single, stout, perfectly healthy and accustomed to eight hours sleep.

Both Cases, 2 and 3, started their sleep-fast at the same time and under the same circumstances and continued it for eighty-eight and one-quarter hours. Unlike Case 1 (which was a preliminary experiment), neither of them experienced any hallucination of vision or any serious suffering. Case 2 was very sleepy the last twenty-four hours. Both slept immediately upon retiring and awoke wholly refreshed, Case 2 after eleven and a quarter hours and Case 3 after fifteen and one-sixth hours. Both subjects gained in weight before sleeping and lost after, or rather during sleep, but, unlike Case 1, their decrease in weight was less than their increase. The observations made were about the same in all cases. According to time measurement Case 2 only found it necessary to make up for sixteen per cent. of his lost sleep; Case 1, twenty-five per cent. and Case 3, thirty-five and three-tenths per cent. It is suggested that this may be accounted for in two ways, viz.: partial sleep during the waking hours or by depth of sleep afterwards. The fact that the subjects all had dreams while awake during the experiments, and the fact also that their sleep was very profound, would indicate that both suppositions accounted for the apparent deficiency. Their urine had an increase of nitrogen and phosphoric acid during the period of insomnia, and relatively more phosphoric acid than nitrogen was excreted. CHRISTISON.

PHYSICAL AND MENTAL MEASUREMENTS OF THE STUDENTS OF COLUMBIA UNIVERSITY. By Prof. J. McKeen Cattell and Dr. Livingston Farrand.

From the *Psychological Review*, Nov., 1896, pp. 618-648.

The authors state, "their chief object in the present paper is the description and discussion of methods rather than the communication of results," and that their object is to "know how a man, for example, has a large head, a short reaction time or a good memory, is likely to vary from the average in other directions, and how likely he is to vary to a certain extent." In this set of experiments they give the

averages of 100 students, 80 being freshmen, and the series contain 10 records and 20 measurements, which were completed within from 40 minutes to an hour; all the recorders had had training in making the tests. About one-half of the students did not vary more than the amount equal to the average of their groups, and these are considered normal. Those above this variation (i. e., twice the average) are considered hyper-normal, and those going below it as sub-normal.

Here is a splendid start towards the construction of a mental criterion much needed, and while the authors evidently comprehend the great importance of their work, they modestly invite suggestions tending to enhance its value in further investigations. Their undertaking so far has been comparatively limited, but they intend to supplement their labors by additional questions conveying almost every point thought to be of possible value, or which in associated relations may help to bring forth a really normal criterion in place of a common one, and reveal more about our common failings as well as defects in our civilization. From this line of investigations we will learn more of our possibilities in nature. But there are several points of inquiry which should be added to the already large lists proposed, and without which some important data will not be secured.

1. The hereditary affections, such as insanity, epilepsy, paralysis, convulsions, asthma, consumption, scrofula, cancer, etc., should be specially inquired into for several generations back.

2. Anything noteworthy in the history of either parents should be mentioned.

3. Remarkable or affecting personal experiences of the subject (student) should be given.

4. His hours of sleep (which in the 24), its quality and mode of onset.

5. Hours and conditions most favorable for study, and how long at a time can he study without fatigue.

6. Habitual dietary: articles particularly liked, articles particularly offensive to nose, palate or stomach.

7. The question of sexual continence or incontinence should also be put in some effectual way since functional impairment of the senses and mental dullness are not infrequently produced by onanism and excessive venery.

8. The hour of the day and the month in which the observations are made, should also be given.

But these questions can only be successfully presented by a shrewd and experienced physician in private. Indeed, the whole investigation should be conducted by a check system, instead of the students' names, and strict confidence the rule. All the delicate inquiries should be made by an outside physician. As this matter is of much interest to neurologists, Prof. Cattell should have no difficulty in finding the medical assistance required.

Students examined 100. Average age, 18 years, and none over 23 years:—

Hair—Black, 8; dark brown, 56; light brown, 34; flaxen, 1; red, 0; not given, 1.

Eyes—Gray, 33; blue, 30; brown, 31; green, 1; not given, 5.

Height—Average in c. m., 175.1.

Weight—Average in Kg., 66.2.

(Both their height and weight are stated as above the average of the population.)

Head—Length, 19.3; v, 0.5; V, 0.2. Breadth, 14.9; v, 0.4; V, 0.1.

Breathing capacity—(98 cases) in litres: 3.73; v, 0.45; V, 0.19.

NOTE—"v" stands for average variation in sets of 10. "V" stands for average variation between sets of 10 and the average of 100.

Vision—Color: (71 cases) 3 per cent. were color blind, and 3 per cent. had defective color vision. Keeness: (94 cases) The right eye

was found to be better than the left. Preference for color: (66 cases) Blue, 34.9 per cent.; red, 22.7 per cent.; violet, 12.1 per cent.; yellow, 7.5 per cent.; green, 6.1 per cent.; white, 6.1 per cent.; no preference, 10.6 per cent.

Hearing—(86 cases) Right ear: normal, 86; sub-normal, 13; abnormal, 1. Left ear: normal, 84; sub-normal, 13; abnormal, 3. In perception of pitch (48 cases), the average variation was nearly one whole tone. 10 per cent. could adjust the monochord within about the one-tenth of a tone; 61 per cent. came between one-tenth and one tone, and 29 per cent. had a greater error.

Dermal sensation was determined by using the æsthesiometer in which the points were 2 c.m. apart and were applied longitudinally on the back of the left hand, between the tendons of the fingers. Five tests were made (49 cases), viz., 2, 2, 1, 1, 2. Correct: 16 per cent., 5 times; 38 per cent., 4 times; 20 per cent., 3 times; 22 per cent., 2 times; 2 per cent., 1 time; 2 per cent., 0 times.

The answers were correct in 67 per cent. of all the trials; 60 per cent. of the cases with two points, and 7.5 per cent. of the cases with one point.

Movement sensation was tested by pulling with a dynamometer three pairs of pulls of about 4 Kg. in strength (48 cases). The average error was 0.63 Kg.

Sensitiveness to pain was tested by an algometer on the ball of right and left thumb (95 cases). Right average: 6.90 Kg.; v, 2.90; V, 0.96. Left average: 6.70 Kg.; v, 2.64; V, 1.94.

Strength of hands was measured by the oval dynamometer; two tests being made with each hand in alternation (99 cases). Right average, 38.9 Kg.; v, 5.7; V, 2.4. Left: 36.4 Kg.; v, 5.3; V, 2.6.

Time Measurement.—The reaction time for sound measured five times in succession with the Hipp chronoscope gave the following results (97 cases). Reaction time: average, 174 ($v=29$); v, 30; V, 13. Time in . Marking 100 A's, mixed with 40 other capitals, gave (93 cases): average, 93 secs.; v, 12.8; V, 6.4.

Average number of A's omitted, 2.6; and seldom a wrong letter was marked.

Perception of Space.—A standard line, 10 c.m. in length, was given on a piece of paper to be copied on another piece (93 cases). Average variation: 6.5; v, 3.4; V, 0.9.

Perception of Time.—A tap began and ended ten seconds, and the student gave a tap when he thought 10 seconds more had passed. Time in seconds: Average, 1.57; v, 0.81; V, 0.20.

Memory.—Three quarters of an hour after the line test for space the student was required to draw from memory another line of the same length (10 c.m.), without previously knowing he would have to do so (21 cases): average error, 7.3 m.m.

After Images.—The student saw in a dark room for 15 seconds a white light of determined area and intensity. Of the 75 students tested, 73.3 per cent. saw an after image, the duration of which was on an average 44.2 secs.; v, 25.2; V, 3.9. Duration of period (latent) between the first and second images was on an average of 16.2 secs.; v, 9.4; V, 6.2. In 61.8 per cent. of the cases the image reappeared in 29.1 per cent. it appeared three or more times; in 7.3 per cent. it appeared four or more times, and in 3.8 per cent. it appeared five times. The after image when first seen was sometimes positive and sometimes negative, and the colors varied greatly, being distributed, in the first place noticed, as follows—negative or dark, 33.3 per cent.; light or white, 29.4 per cent.; blue, 13.7 per cent.; purple, 9.8 per cent.; green, 5.9 per cent.; yellow, 3.9 per cent.; red, 2.0 per cent.; miscellaneous, 2.0 per cent.

Imagery was tested by letting the students fill in a blank containing the questions below (95 cases): Think of your breakfast table

as you sat down to it this morning. Call up the appearance of the table, the dishes and food on it, the persons present, etc.; then write answers to the following questions—

1. Are the outlines of the objects distinct and sharp? Yes, 86.5 per cent.; no, 6.2 per cent.; miscellaneous, 7.3 per cent.
2. Are the colors bright and natural? Yes, 83.3 per cent.; no, 10.4 per cent.; miscellaneous, 6.3 per cent.
3. Where does the image seem to be situated? In the head? before the eyes? at a distance? In the head, 28.7 per cent.; before the eyes, 36.2 per cent.; miscellaneous, 2.1 per cent.
4. How does the size of the image compare with the actual size of the scene? Same, 53.7 per cent.; smaller, 45.3 per cent.; miscellaneous, 1 per cent.
1. Can you call to mind better the face or the voice of a friend? Face, 75 per cent.; voice, 14.6 per cent.; miscellaneous, 1 per cent.
2. When "violin" is suggested, do you first think of the appearance of the instrument or the sounds made when it is played? Appearance, 76.8 per cent.; sounds, 23.2 per cent.
3. (a) Can you call to mind natural scenery, so that it gives you pleasure? (b) music? (c) the taste of fruit? Scenery: Yes, 94.6 per cent.; no, 4.3 per cent.; miscellaneous, 1.1 per cent. Music: yes, 89.1 per cent.; no, 9.8 per cent.; miscellaneous, 1.1 per cent. Taste of fruit: yes, 68.1 per cent.; no, 28.6 per cent.; miscellaneous, 3.3 per cent.
4. Have you ever mistaken a hallucination for a perception, e. g., apparently heard a voice or seen a figure when none was present? If you answer yes, describe the experience on the back of this sheet. Yes, 74.7 per cent.; no, 25.3 per cent.

The students have promised to submit to a re-testing once a year.
CHRISTISON.

NEURO-PATHOLOGY.

THE CONDITION OF THE TENDON REFLEXES IN TRANSVERSE MYELITIS. Wiener klinische Wochenschrift, Oct. 1, 1896. By Ernst Bischoff.

It has been known for some years that the patellar reflex may be absent when the spinal cord is completely destroyed in the cervical and upper thoracic regions (Bastian), and no satisfactory explanation has ever been given. Dr. Bischoff has examined the reported cases in which this condition had been present, and he concludes from the study of these that the reflexes may be lost when the interruption of cord fibres is incomplete, and may be preserved when the destruction of the fibres in *loco morbi* is total. The condition of lessened reflexes, according to him, is due to some process which has no direct connection with the spinal lesion, and is not dependent on its completeness or height, nor on the degree of the secondary degeneration.

His explanation is as follows: After the rapid formation of a partial or complete transverse lesion in the cervical or thoracic cord (with the exception of its lowest portion) the vasomotor nerves which supply the abdominal viscera and the lower portions of the body, together with the motor and sensory fibres, are paralyzed. This causes dilatation of the blood vessels in these parts and a decrease in the general blood pressure. The lumbar region of the cord suffers from the anæmia thus produced; its vitality is lessened, and the tendon reflexes are lost.

Acute decubitus, vomiting, diarrhœa, albumenuria, nephritis and occasional œdema of the legs, seen in such a condition, are the results of these vascular changes.

The vasomotor disturbance is only temporary, and if no organic changes have occurred, the reflexes may be restored.

The higher the spinal lesion is situated, the greater must be the vascular disturbance, as more fibres are destroyed.

In most cases the tendon reflexes have not returned after they have been once lost. In some of these there has been a general septic infection, with visible organic changes in the muscles and nerves of the lower extremities or in the lumbar cord; in other cases, rapid death has prevented the formation of these visible alterations. Where the loss of the tendon reflexes after the formation of a transverse lesion has been gradual, the cause is to be sought in secondary morbid processes; as extension of a tubercular meningitis; neuritis due to alcohol, toxæmia or cachexia; or general septicæmia, which cause degeneration of nerves and muscles.

Dr. Bischoff acknowledges that his explanation is entirely hypothetical. SPILLER.

FLACCID PARAPLEGIA WITH ANÆSTHESIA, COEXISTING WITH EXAGGERATED KNEE-JERK, FOOT CLONUS AND SPASM OF THE BLADDER AND RECTUM.

The rarity of the above syndrome induced Dr. Journier (Lyon Med., June 28, 1896) to publish his observations and the resulting conclusions: The patient, thirty-four years old, had no positive previous history. His present trouble began three months ago with pains in the lumbar regions, then radiating pains around the abdomen, then progressive paraplegia set in, which has developed so rapidly, and to such an extent, within the last two months, that the patient is now powerless. There were concurrent vesico-rectal troubles. The examination revealed further: Pains on pressure over the second and third lumbar vertebræ; flaccid paraplegia with absolute loss of power of both legs; complete anæsthesia in this region with a zone of hyperæsthesia above the glutei; slight œdema of the feet; Laseque's signs of both sciatic nerves; exaggerated knee-jerks, and well marked clonus of the feet; exaggerated cutaneous reflexes, but less marked; desire to micturate frequently, but unable to satisfy it without passing catheter, which reveals spasms of the sphincter; spasms of the sphincter ani, and, in order to help defecation, the patient is obliged to introduce a finger into the anus. The diagnosis of Pott's disease, with compression of the lower lumbar cord and terminal cone, was made by Professor Lepine. The author remarks that the tendon reflexes are considered in general to depend entirely upon the condition of muscular tone, but the present observation goes to prove their independence. Accepting the theory of the tendon reflex centres as being in the spinal cord, the phenomena might be explained as follows: The compression of the spinal cord by the diseased vertebræ did not produce very extensive lesions in its substance, but these lesions might easily be of sufficient intensity to be annihilatory, inhibitory, or destructive at a certain point—for instance, in the upper portions of the conus terminalis, and adjacent cord—and only sufficient to be stimulating higher up, or perhaps lower down. The centre for the patella tendon reflex is usually localized by Starr in the extremity of the conus terminalis. These are all distinct centres, although they are connected with the spinal centres for movements of the lower extremity, the bladder and rectum. If, notwithstanding the complete anæsthesia and paralysis, percussion of the patella tendons causes the foot to be thrown forward forcibly, it is because the crural nerve contains special sensory fibres. These fibres transmit impulses through a fascicle of collateral fibres, described by Ramon y Cajal, to be a special group of cells in the anterior horns, which are therefore motor reflex cells. The same interpretation might be applied to other reflex movements, as for instance, the foot clonus. The flaccidity and the exaggerated reflex motility of the muscles could not be accounted for by the hypothesis of a transverse myelitis with compression of the pyramidal tracts. If muscular tone, ordinary and reflex movements, were controlled by the same anterior spinal neuron, one would expect

similar disturbances of these three functions. But the foregoing authentic example of disassociation, the author thinks, should justify his advancing the theory of different motor neurons in the anterior horns, the ones controlling reflex movements, the others voluntary motility and muscular tone, both of which, however, are connected with each other by the pyramidal fascicle of the cerebral cortex. Therefore, the hypothesis of the existence of a peripheral neuron in a spinal ganglion, the function of which is transmission of peripheral impulses to a motor reflex neuron, would not seem unreasonable, and might help explain the symptoms of the pseudo-tabes of peripheral origin.

MACALESTER.

PARAPLEGIA: GUNSHOT WOUND OF THE SPINAL CORD. Whittaker, Rausohoff and Kramer. *International Medical Magazine*, November, 1896.

Under the above title, Whittaker embraces the occasion offered by an interesting clinical case to speak at some length upon lesions of the spinal cord. The cited case offered many peculiar features, both in symptomatology and treatment. A young married woman had five weeks previously accidentally shot herself through the abdomen. The pistol ball passed through the stomach and lodged in the vertebral column. Collapse and blood vomiting followed immediately, but no loss of consciousness occurred. The lower part of the body became instantly paralyzed. Sensation to touch and pain lost below the stomach line, bowels and bladder functionless. In a few days bed sores began to develop and great pain was felt in the upper part of the body. Status at time of admission: lower half of body markedly atrophic; paraplegia complete and absolute; all reflexes abolished; skin white and scaly; large, deep bed sores over the sacrum, trochanters, knees and ankles; cicatrix of the entering bullet in the median line, some inches below ensiform process. Diagnosis: Wound of spinal column with pressure on the cord. Treatment: Euphraphin paste on bed sores; catheterized; bowels flushed; placed on water-bed.

By means of X-rays the bullet was located in or near the eighth dorsal vertebra. Operation revealed its lodging-place there in the body of the vertebra. The cord was compressed by protruding bone at that point (eighth dorsal), and seemed considerably broken up.

After the operation the patient slowly rallied, but the bed sores persisted and the patient was placed in the water bath at 90° F. She was taken from the tank only for an hour a day and placed on a cot. Under this treatment improvement began and the bed sores commenced healing. Morphine reduced to four grains in the twenty-four hours. Whittaker's discussion of the case was aided by "Remarks on Surgery of the Spinal Column and Cord" (Rausohoff) and "Explanations on the Use of X-Rays in Diagnosis" (Kramer, who took the skiagraph). Taken altogether the subject was most interestingly treated. Rausohoff and Kramer warn not to make a too hopeful prognosis in spite of the successful operation. The remarks of the former, a noted surgeon, were especially noteworthy, as he maintains the lesion could readily have been located without the use of the X-rays. Whittaker, being intimately acquainted with the physiology and pathology of cord lesions, seems to take no very hopeful view of our efforts to rescue cases of cord lesions. Regeneration of the spinal tissue may perhaps be possible, but no sure signs are as yet known which would lead us to believe in such a possibility. The above article is of such importance and so clearly dealt with, that it should be read carefully in the original, notably on account of the prognostic conclusions.

STERNE (Indianapolis).

POTT'S DISEASE WITH EARLY ANÆSTHESIA.

Chipault (*La Médecine Moderne*, Aug. 15, 1896) describes a case

which he believes to be unparalleled, in the appearance of objective sensory symptoms before any motor troubles, a reversal of the ordinary course of such cases. The patient, eleven years of age, was received in the hospital complaining of pains in the back and about the waist. On a careful examination it was found that there was complete cutaneous anæsthesia and analgesia as high as the middle of the thighs. There was no difficulty of motion except that station was bad with the eyes closed. It was at first supposed that the trouble was hysterical, but two months after the first examination the patient was brought back with all the symptoms—vertebral, motor and sensory—of a serious paraplegia. The anæsthesia had increased and now extended as high as the umbilicus. The patient died of bronchial pneumonia a few days later, but no autopsy was permitted. MITCHELL.

EXAGGERATION OF KNEE-JERK IN ATHEROMATOUS PATIENTS.

M. F. de Grandmaison (*Médecine Moderne*, Dec. 23, 1896) has studied in twenty-six cases of atheromatous patients, carefully excluding those with disseminated sclerosis or medullary syphilis, the condition of the knee-jerks. Of fourteen men, three cases gave no evidence of medullary trouble, the others all had the reflexes manifestly exaggerated, and ten of them exhibited the phenomena of spinal epilepsy; four showed merely a tremulousness, three a slight clonus and three a very marked clonus.

Of twelve atheromatous women, three had no evidence of spinal trouble and nine had a marked exaggeration of the patellar reflex. Of these nine, five had very slight clonus, one more marked, and in three it was very decided.

Reduced to percentages, this would show that 78 per cent. of the men had some disorder in the spinal cord and 75 per cent. of the women. De Grandmaison concluded that spinal atheroma is less often diagnosed than similar cerebral conditions, because troubles of motility, if slight, are less noticeable than those which affect the intelligence.

MITCHELL.

SPASTIC PARAPLEGIA IN SISTERS.

MM. Achard and Fresson report in the *Gazette Hebdomadaire*, Dec. 24, 1896, two cases of spastic paraplegia in sisters. The first patient was the second of eleven children. The disease showed itself at the age of one year, after an attack of variola. Previous to this she had walked well. She presented all the characteristics of the trouble: contractions, with rigidity of the legs, exaggerated knee-jerks, bad station; no trophic or sensory disorder and ordinary good general health. The second sister, the ninth child of her parents, at the age of ten months, had an acute illness of doubtful character before she had begun to walk. At the age of sixteen months troubles in walking were marked; upon her first attempts at movement she walked upon the point of the toes, dragging the toes along the floor. This grew no worse until about the ninth or tenth year, from which time it was progressively aggravated, until at the time of study of the patients, her condition was like that of her sister, but not quite so marked. The family history was in no way remarkable except for the neuro-pathic taint indicated by the presence of epilepsy in another sister. The unusually early appearance of the first symptoms in these two patients is of interest; no cases have previously been reported at such an age.

MITCHELL.

GEHIRNBESUND BEI SPASTISCHER LAEHMUNG MIT MUSKELSCHWUND.

[Cerebral Findings in Spastic Paralysis with Muscular Atrophy.]
Wiener klinische Wochenschrift, No. 45, 1896. By G. Anton (Graz).

Among the most interesting features of the case reported in this paper were: weakness and wasting in the muscles of the hands and

forearms, followed in about six months by dysphasia, dysphagia and salivation. When the patient was examined about eight months later, atrophy in the muscles of the face and tongue, inability to close the mouth voluntarily, exaggerated chin jerk, atrophy *in toto* of the muscles of the upper limbs, increased reflexes, spastic paresis in the legs, and reaction of degeneration were observed.

The anterior roots as well as the cells of the anterior horns throughout the cord, but less in the lumbar region, were degenerated. The pyramidal tracts were also degenerated. The hypoglossal nuclei and nuclei of the motor trigeminal nerves were atrophied. The degeneration of the pyramidal tracts was traced to the cortex. There was a decrease in the number of large cortical ganglion cells.

The case was one of amyotrophic lateral sclerosis. It is not probable that the disease began primarily in the cerebral cortex; it is more likely that both central and peripheral motor neurons were affected by a common cause.

SPILLER.

INSULAR SCLEROSIS AND HYSTERIA. Thomas Buzzard. *Lancet*, Jan. 2, 1897.

In a clinical lecture Prof. Buzzard calls attention to the difficulties in the differentiation of insular sclerosis and hysteria. Aided by the demonstration of two cases of insular sclerosis and the citation of several others, he points out the special symptoms particular to the two maladies in question, emphasizing the necessity for closer study of the incipient stages of insular sclerotic disease. In almost every case of beginning organic affections of this class, the diagnosis, "Hysteria," is usually made, and this semblance to the functional disease is very often marked. However, it should, if possible, be excluded; and he, therefore, points out certain characteristics which his large experience has taught him to value highly, though he is careful to claim no pathognomonic significance for them. These special signs he considers as follows:—

1. Intention tremor, if well marked, appears to hold an important place in the diagnosis of insular sclerosis. A certain clumsiness upon intended movements, probably due to a loss of the muscular sense, is frequent in hysteria, but well-marked "intention tremor" must be very rare in functional disease.

2. Nystagmus or nystagmiform movements must also be considered important evidence of organic trouble.

3. Ankle clonus is a very valuable symptom of an affection of the antero-lateral tract of the cord. It occurs sometimes in functional diseases, but is not usually pronounced. If ankle clonus be well marked, organic disease is nearly always present.

4. Atrophy of the optic disc is a symptom of greater value than any of the foregoing. When it is present, it bears strong proof of organic lesion. Pallor of the disk and narrowing of the concentric visual fields will be found almost invariably associated with some visual defect. The fact that concentric narrowing of the visual fields is frequently present in hysteria renders the differential diagnosis more difficult, but it is probably always unassociated with changes in the appearance of the disc.

5. To the state of the plantar reflex Buzzard ascribes considerable value. In hysteria he has found it almost invariably changed, namely either entirely absent or only feebly expressed. When an hysterical paraplegia disappears the plantar reflex usually returns. The retention of the plantar reflex in a case of loss of power in the legs is strong presumption that structural changes are present. The converse, however, does not apply.

Lastly, Buzzard considers it unsafe in a doubtful case to make a diagnosis of insular sclerosis, until such symptoms as nystagmus,

pallor of the optic disk, marked intention tremor, or incontinence of urine make their appearance.

"But in a case in the history of which paresis has occurred in one or more limbs, with spontaneous recovery, and repeated recurrence of the symptom in the same or in another part, after longer or shorter intervals, together with a history of amblyopia, either temporary or persistent, my experience would lead me to diagnose insular sclerosis in the absense even of the symptoms first enumerated."

The last sentence is quoted verbatim in order to give Prof. Buzard's exact expression, for this picture appears frequently enough in hysterical subjects to allow some doubt as to its absolute application to organic disease.

STERNE (Indianapolis).

A CASE OF ENDOTHELIOMA OF THE CEREBRAL MEMBRANES WITH JACKSONIAN EPILEPSY AND WASTING OF THE PARALYZED MUSCLES. Archives of Pediatrics, September, 1896. By Frederick A. Packard.

The atrophy in the deltoid, thenar and hypothenar eminences, and interossei muscles of the left upper limb, similar to that seen in chronic anterior poliomyelitis, made this case of much interest. The left arm was flaccid and motionless, the left leg was absolutely powerless. The deep reflexes were absent in all the extremities. The symptoms presented by the patient were those of brain tumor.

At the autopsy a mass 10 c. m. long was found in the parietal and posterior portion of the frontal region on the right side, extending outward 5 c.m. from the median line, and downward 4, 5 c.m. on the median surface in the great longitudinal fissure.

Dr. Packard believed the atrophy of the left arm was undoubtedly due to central disease. Inasmuch as the arm was violently exercised by the convulsive movements of the patient, the atrophy could not be attributed to disuse. The appearance of the arm also was unlike that seen in atrophy from disuse. The motor tracts in the pons, oblongata and upper part of the cervical cord, were normal.

Neuritis was excluded on account of the absence of sensory symptoms. The first signs of trouble with the left arm occurred only five months before atrophy in marked degree was noticed.

Unfortunately the cervical swelling of the cord could not be obtained, and the electrical reactions were not known.

SPILLER.

BEITRAG ZUR DIAGNOSTIK UND ZUR CHIRURG. BEHANDLUNG DER GEHIRNTUMOREN UND DER JACKSON'SCHEN EPILEPSIE. [A Contribution to the Diagnosis and Surgical Treatment of Brain Tumors and Jacksonian Epilepsy.] Deutsche Zeitschrift für Nervenheilkunde, Band 9, Heft 3 u. 4. By Fr. Schultze.

The following symptoms were observed in a certain case: Bilateral ophthalmoplegia externa, with little involvement of the sixth nerves, ataxia, dysphagia, dysphasia, moderate exaggeration of the tendon reflexes in the lower limbs, disturbance of the vesical functions, choreiform movements of the lower extremities, intense apathy, and swelling and reddening of the papillæ. In the beginning of the disease paræsthesia was complained of in both hands and feet.

The symptoms seemed to point toward tumor in the region of the corpora quadrigemina, and were very similar to those observed in a case of tumor of this region reported by Bruns.

In the course of some months the symptoms entirely disappeared. This rendered the diagnosis improbable. Schultze regards the case as one of polioencephalitis and ependymitis, with symptoms of pressure from hydrocephalus of a mild grade. Some little time later a psychosis developed. Siemerling has observed psychoses associated with ophthalmoplegia very frequently.

In the second case the diagnosis of brain tumor was correct, but

the location was only in part what was supposed. As the pain at first was more on the left side, and the left abductens were weaker than the right, the pressure was supposed to be chiefly in the left hemisphere. Later the right half of the body was paralyzed.

The tumor was found in the lower half of the cerebrum, but chiefly on the right side. It is possible that pressure was made by the tumor on the left internal capsule, although no evidences of this were present at the autopsy. It was remarkable that there were no symptoms of pressure on the right internal capsule. The left lateral ventricle was much dilated, perhaps this may be regarded as an explanation of the right sided paralysis. The right lateral ventricle was almost closed by the tumor. Decussation of the pyramids was present.

The diagnosis of tumor in the third case was correct, and the growth, though large, was easily removed, but the patient, a vigorous young man, died a few hours after the operation.

In the fourth case the diagnosis of tumor was also made. The only focal symptoms were partial paralysis of the muscles supplied by the right facial nerve, with occasional paresis of the right arm. Cerebellar gait had been noticed. At the autopsy a large tumor was found in the left facial centre, which had caused displacement of the surrounding tissue. A cyst was found in the medullary substance somewhat more anteriorly. No distinct attacks of Jacksonian epilepsy had been noticed. The partial facial monoplegia was striking, in view of the large size of the tumor.

In the fifth case the following symptoms were noted: Rigidity of the neck, headache in the frontal and occipital region, hyperalgesia of the muscles of the legs, vomiting, frequent yawnings, papillitis in both eyes, rhythmical contractions of the right sterno-cleido-mastoid, apathy, paralysis of the right external rectus with weakness of the internal.

The operation revealed a tumor of the right frontal lobe. The patient died soon after the operation.

Schultze believes the operation of cranial resection is not without risk. The lowering of pressure in the cranial cavity after removal of a large tumor is a source of danger.

In two cases of Jacksonian epilepsy, reported by Schultze, operation gave no permanent relief. SPILLER.

A CONTRIBUTION TO THE STUDY OF JACKSONIAN EPILEPSY. *Lancet*, Oct. 24, 1896. By W. J. Harris.

The first case shows that certain movements of the abdominal muscles are unilaterally represented in the cerebrum; while the second demonstrates the unilateral cortical representation of the soft palate and tongue movements (Beevor and Horsley). A child of five years, with symptoms of tuberculous tumors in both Rolandic regions, and in the cerebellum, had five attacks of convulsions while in the hospital, three right-sided and two left. In the attacks the abdominal muscles of the side affected were thrown into spasm, and the head and eyes were turned to the same side. The opposite sterno-cleido-mastoid muscle was, therefore, contracted. Movements, not muscles, are represented in the cortex. The sterno-mastoid, when employed to turn the head to one side, is governed by the hemisphere opposite to the side toward which the head is turned.

Case II.—This patient had visual attacks, in which he saw red and green lights, usually accompanied by an olfactory sensation of a "nasty, burning smell," and sometimes by a bitter taste in the mouth. These symptoms had followed a fit he had had six months previously, which left him weak on the left side with almost complete left hemianopsia. The patient had had syphilis. A left-sided convulsive attack, in which there was involvement of the left side of the tongue and soft palate, was observed.

Dr. Gowers diagnosed chronic meningitis of the convexity, attacking especially the angular and motor gyri.

At the autopsy the pia was found to be adherent over the lower part of the right ascending parietal and ascending frontal regions. There were localized patches of softening in the right angular gyrus, on the median surface of the right occipital lobe, in both uncinate gyri, in the depth of the parieto-occipital fissure, and just in front of the internal parieto-occipital fissure in the precuneus.

The writer states that the softening in the right cuneus accounted for the left hemianopsia and possibly also for the visual attacks, the softening in the uncinate gyri for the olfactory sensations, while the thickened and adherent membranes over the lower ascending frontal and parietal convolutions explained the localized convulsions in the left side of the palate, tongue, face and hand. SPILLER.

A CASE OF DYSLLEXIA: A PECULIAR FORM OF WORD-BLINDNESS. *Lancet*, Nov. 21, 1896. By James Hinshelwood.

The patient described in this paper on attempting to read printing or writing could read the first few words quite correctly, and would then suddenly come to a stop, saying he could not go on. After resting a little he would make a further attempt with precisely the same result. He stated that, although he could see the letters, they seemed to lose all meaning for him, that they were not blurred, however, and did not seem to run together. Visual acuity was unimpaired. The patient had previously been a good workman, but he seemed to have forgotten all methods of work, and had to be shown every successive step. He was unable to recollect after a few minutes where he had placed any article. He frequently lost his way. A careful examination of the nervous system revealed no abnormality. His eyes were normal, with the exception of a slight amount of presbyopia. There was no general deterioration of the mental power. His memory for past events was not impaired. There were no disturbances of speech.

He was told to practice reading a short time every day, and when he left the hospital, after seven weeks, he could read for any length of time. His other symptoms improved, and he soon became almost entirely well. The writer regards this improvement chiefly as the result of the withdrawal of alcohol.

Dr. Hinshelwood believes, if the visual memory centre be itself intact, and the conductivity of the connecting fibres be only partially impaired, there may not be absolute inability to read (alexia), but there may be very great difficulty in interpreting written or printed symbols (dyslexia).

Berlin regards dyslexia as a special form of word-blindness due to an interruption in the conductivity of the fibres to the visual centre in the lower parietal lobe of the left hemisphere, and he states that post-mortem examinations have shown that the anatomical seat of the lesion in dyslexia is to be found in the lower parietal lobe of the left hemisphere, which includes the supra-marginal and angular convolutions. Berlin has found dyslexia as a temporary symptom in chronic alcoholics. It has also been observed as the first symptom of grave organic disease of the brain.

Dr. Hinshelwood believes his case was of toxic origin, and that the peculiar forgetfulness was due to a failure of visual memory, the centre for which is in the supra-marginal and angular gyri, as is also the centre for word-seeing. Word-blindness and loss of memory for places and objects are varieties of mental blindness. In complete mental blindness, involving all forms of visual memory (including word-blindness), there is probably a bilateral lesion in the centre of both sides of the brain.

The author believes that in his case there was impairment of the

functional activity of the right as well as of the left centre, especially as many of the nervous disorders due to alcohol are bilateral.

[If word-hearing were normal, and spontaneous writing were possible in this case, there might be some doubt as to the propriety of locating the lesion within the speech zone of the left cortex.]

SPILLER.

THE SIGNIFICANCE OF HERPES LABIALIS IN THE DIFFERENTIAL DIAGNOSIS BETWEEN SUPPURATION AND TUBERCULOUS MENINGITIS. From Prof. Eichhorst's Clinic, University of Zürich.

Dr. A. Habel (*Deutsche Med. Wochenschr.*, Oct. 15, 1896) publishes a case of tuberculous meningitis in a young woman that ran subacute course, during which herpes labialis developed. Lumbar puncture was made but no fluid could be aspirated. The conclusions reached are: I. Herpes labialis is a rare symptom in tuberculous meningitis, but by no means excludes it, as maintained recently by F. Klemperer. II. Lumbar puncture is, in most cases, an excellent means of diagnosis, but frequently fails to reveal tubercle bacilli, and even fluid in the dura sack. III. In the aspirated fluid, mucous coagulations are found that indicate the tuberculous nature of the disease.

MACALESTER.

UEBER MENINGITIS SEROSA UND VERWANDTE ZUSTANDE. [Concerning Meningitis Serosa and Related Conditions.] *Deutsche Zeitschrift für Nervenheilkunde*. Band 9, Heft 3 u. 4. By H. Quincke.

Quincke reports a number of new cases of this form of meningitis, which at the autopsy usually presents no notable findings. He calls attention to the difficulty of making a diagnosis, which may be lessened by the lumbar puncture. The exudation in many cases is like that seen in joints, and in the acute circumscribed oedema of the skin and mucous membranes. It is probable that there are gradations from the purely physiological to the inflammatory exudations. It is not unlikely that in severe forms of migraine an actual meningeal exudation is present.

He recommends the use of mercury and the salicyates for the serous meningitis. Lumbar puncture relieves the pressure.

SPILLER.

EIN FALL VON POLYNEURITIS MIT MULTIPLN SCHWIRLERNARTIGEN GRANULATIONS-GEWÜLSTEN DER HAUT (A Case of Polyneuritis with Multiple Callous Granulomata of the Skin). *Deutsche medizinische Wochenschrift*, No 45, 1896. By A. Fraenkel.

Fraenkel reports a case of multiple neuritis, in which the left facial nerve was also affected, with dermal tumors in the extremities. These developed in the beginning of the disease and were symmetrically arranged. Histologically, the tumors resembled the granulomata. They consisted of small round cells, large epithelioid cells, spindle-shaped and giant cells. As bacilli could not be demonstrated and inoculation gave negative results, the process was not regarded as tubercular. The patient denied syphilitic infection and presented no signs of the disease. Gummata of the skin, if not cured by anti-syphilitic treatment, usually soften and ulcerate, or more rarely contract in consequence of central caseation. In this patient contraction of some of the tumors was observed as long as the treatment was continued, and the contraction involved the whole of the growths. There was no epidermal desquamation and no pigmentation. It has not been positively shown that syphilis causes multiple neuritis. The patient had been exposed to lead poisoning. The facial paralysis, the severe paræsthesia and spontaneous pain, the sensitiveness to pressure of the nerves, skin and muscles, are difficult to explain as symptoms of lead intoxication. Facial paralysis is rare, and granulomata of the skin seem to be unknown in saturnine poisoning. It is possible that the

tumors were of a rheumatic origin. They were probably due to the poison which caused the neuritis. Fraenkel acknowledges that he is unable to state the nature of these growths, but is inclined to attribute them to syphilis. SPILLER.

PERIPHERAL NEURITIS IN PHTHISIS.

It has been known for some time that peripheral neuritis may complicate pulmonary tuberculosis. Not long since Carriere, of Bordeaux, published a thesis on this subject and he now adds two new cases (*Arch. Clin. de Bordeaux*).

The first case was a woman of thirty-five, whose tubercular trouble began in the intestines but soon involved the lungs also. About a month after the invasion of these organs the patient began to have almost continuous lancinating pains in the lower extremities, worse in the popliteal region and calves, with great tenderness of the sciatic nerve and leg muscles and hyperexcitability of the latter. There was some atrophy, corresponding weakness, and almost a disappearance of the knee-jerks. Sensation was normal, but the skin showed slight trophic changes. Soon after the beginning of the symptoms just detailed exquisite "dermalgia" appeared; the patient could not endure the weight of the bed-clothing.

The pulmonary and peripheral diseases advanced, the muscles of the lower extremities became atrophied to a marked degree and exhibited reaction of degeneration, all reflexes were lost and the patient became completely paraplegic. She died one and a half months after the inception of the neuritis.

The autopsy was very carefully made and included a thorough microscopic examination of the nervous system. There was advanced pulmonary tuberculosis. All the nerves below the knee were completely degenerated or very nearly so. The sciatics at the notch were practically normal. Spinal cord, spinal meninges, lumbar nerve roots and nerves of the upper extremities were normal.

The second case was one of pulmonary tuberculosis, following pleurisy, in a man of fifty-one. When first seen, about the only indication of neuritis was atrophy of the small muscles of the right hand, especially the thenar eminence; feeble knee-jerks and occasional lancinating pains in the thumb and first two fingers of the right hand were present. There was in addition some appearance of glossy skin on the same hand. While in this condition the patient suddenly died of hemoptysis. In this case also careful post-mortem examination was made, which showed the nerves of the lower extremities and those of the left upper extremities to be perfectly normal. On the right side the median nerve above the elbow showed very slight changes, at the wrist marked degeneration, and in the branches going to the thenar eminence the nerve fibres had entirely disappeared. The ulnar nerve was normal. As in the former case, the brain, spinal cord, membranes and nerve roots showed no change.

Clinically, the cases are of interest as showing the marked difference in the distribution of the neuritis and in the symptomatology of the same. Cases like the second, in which atrophy of the small hand muscles is the principal symptom, are exceedingly rare. The author considers at some length the *modus operandi* of tuberculosis in causing these cases of neuritis. Basing his conclusions on his own post-mortem examinations and experiments on animals, he proceeds to exclude as a cause congestion of the thoracic and spinal vessels (Leudet), purely functional disease (Weill), cerebral lesions (Blocq and Marinesco), spinal meningitis (Arthaud and others) and an affection of the cells of the anterior horns of the cord (Erb). This exclusion is, we think, quite justified, as the microscopic examination was made with the latest and most approved technique. He excludes also inanition as a cause, as the patients ate well, and, besides, guinea pigs that he starved to death never showed peripheral neuritis.

The degenerated nerves were also carefully examined for tubercle bacilli with negative results and four guinea pigs were inoculated according to accepted methods without effect. The conclusion seems inevitable that the neuritis could not be due to the presence of bacilli in the nerves themselves.

Apparently the only hypothesis left is that of the author, that the peripheral neuritis of phthisis is "due to the action of the tubercular poison," which he considers identical with the toxins "secreted by Koch's bacillus."

It should be remarked, however, that the absence of a tuberculous process in the nerves in these cases does not preclude the possibility of its presence in others. Indeed, we should expect occasionally to find tubercles in the nerves as well as other tissues of the body.

PATRICK (Chicago).

THE CENTRAL NERVOUS SYSTEM IN POLYNEURITIS. Du Systeme Nerveux Centrale dans la Polyneurite. S. Soukhanoff. Archiv. de Neurologie, March, 1896.

After a cursory review of previous observations upon central lesions found in multiple neuritis, the author gives the clinical history of an aggravated case of alcoholic neuritis occurring in a patient of twenty-seven years of age, the alcoholic habit extending over a period of eight years, the neuritis itself being of two year's standing.

The microscopical examination of the peripheral nerves showed a diffuse parenchymatous neuritis. The spinal cord was investigated by the method of Marchi and showed in the lumbar region marked degeneration of the posterior median and posterior lateral columns, the degeneration being more marked in the outer border of Burdach's columns and Lissauer's marginal zones. In addition, there were a few fibres of degeneration in the anterior and lateral columns. In the dorsal region of the cord the lesions were similar, but were more equally distributed, the columns of Goll and Burdach being involved in an equal degree. The cervical region showed marked degeneration of the columns of Goll with a few fibres degenerated in Burdach's column. Advancing into the pons, the lesions were most marked in the nucleus gracilis, the degenerated fibres being clearly traced to this nucleus and ending there. The nucleus cuneatus was involved in a very minor degree and a very few fibres in a state of degeneration were found in the hypoglossal, facial, abducens and oculo-motor nerves.

JELLIFFE.

POST-TYPHOID NEURITIS.

After enumerating the various causes which are apparently responsible for the production of neuritis, Dr. George J. Preston (Maryland Medical Journal, Vol. XXXVI., No. 4) calls attention to typhoid fever as a rare but unmistakable factor in the etiology of that disease. In confirmation of this fact he cites three interesting cases which came under his observation.

Case I.—A young man of twenty-four was taken with a typical typhoid. The fever lasted from May 30 to July 21, which time includes a relapse. With the beginning of convalescence the patient began to complain of pain in the legs, the slightest contact with the bedclothes produced great suffering. For three or four days there was an erysipelatous blush over the right leg, and later a small abscess developed over the ankle. The pains in the legs continued and an examination revealed loss of patellar tendon reflex, some atrophy, double-foot drop and reaction of degeneration. There was no marked disturbance of sensation.

Case II.—Young woman. The typhoid was severe and protracted. Four days after the subsidence of the fever the patient complained of intense pain in the right arm and leg, with inability to move them. In ten days the pain went away, but came back with increased severity

after spending a day in the country. This time the whole body was affected, but most excruciatingly in the left leg. Besides the pain, the following symptoms were present: loss of patellar reflex, muscular atrophy, reaction of degeneration in both lower extremities and double foot drop. Very little loss of sensation and no disturbance of bladder or rectum.

Case III.—Patient tubercular, aged twenty-eight. The disease ran the usual course of about five weeks. Bed sores developed and the temperature assumed a septic type. The patient developed intense hyperæsthesia, most marked in the lower extremities. Paralysis of the extensor muscles of both arms and legs appeared, followed by atrophy. A gradual return of power occurred, though the paralysis never completely disappeared.

ABRAHAMS.

ABSENCE OF TENDON-ACHILLES REFLEX IN SCIATICA.

At the Soc. Med. des Hôp. (Médecine Moderne, December 23, 1896) M. J. Babinski reported that he had observed in several cases of sciatica an absence of the usual reflex of the tendo-Achilles, or a comparatively great weakness of this reflex upon the affected side. The phenomena was found not only in intense sciatica with atrophy and the presence of sciatic neuritis, but also in the more purely neuralgic forms of the affection. He presented a man to the society in whom the disease had lasted for a year with sufficient severity to have given rise to the form of sclerosis described by Charcot and Babinski, and stated that at one time abolition of the reflex had been almost complete, but that since the patient's improvement it had returned, although still less marked than on the opposite side.

While this phenomenon is not of great importance from a diagnostic point of view, it may be useful to assist in the differentiation of true sciatica from hysterical forms of the disease in which the reflex will probably be found normal; although B. says he "he can find no reference to it in the text-books," the reporter has seen it made use of in this country, as well as the similar decided reduction in the knee-jerk found in sciatica.

MITCHELL.

LINGUAL HEMIATROPHY.

Dr. Babinski reported at the Medical Society of the Hospitals (Medical Week, Nov. 27, 1896) a case of hemiatrophy of the tongue, consecutive to a lesion of the hypoglossus, in a patient suffering from tubercular osteo-arthritis of the left occipito-atlanteal articulation.

When the mouth of the patient was half open the tongue occupied the middle; but when it was widely open, with the tongue drawn back, the tip deviated toward the healthy side. On the contrary, when the patient put out the tongue, it deviated toward the side of the hemiatrophy.

J. K. MITCHELL.

OPHTHALMOPLAGIC MIGRAINE.

In a Paris thesis Dr. Alché (Gazette Hebdomadaire, Dec. 10, 1896) has collected all the cases of this disorder, amounting to twenty-five, which have hitherto been reported. Periodic oculomotor paralysis is rather a complex of symptoms than a morbid entity, and sufficiently defined by Charcot as a "hemisideria with the necessary accompaniment of the total paralysis of one of the oculomotor nerves." While its etiology is still obscure, the observation in Europe has been that, contrary to the occurrence of ophthalmic migraine, it is most common in the working classes and in the years of early adolescence. The headache, its first symptom, all authorities are agreed in regarding as characteristically unilateral but somewhat more diffused than that of ordinary hemisideria. Its seat of preference perhaps is in the neighborhood of the eye-brow, but it extends to the frontal and temporal region, sometimes to the occiput or the nape, sometimes to both. Ballet has observed its radiation into the eyeball. It is usually not ex-

cessively severe, but rather a dull, deep ache, with exacerbations, sometimes in the morning and sometimes in the evening.

Oculomotor palsy occurs at a variable time after the appearance of the pain. It is always unilateral and quite complete, affecting only the side upon which the pain is seated. The most peculiar characteristic of the disorder is its tendency to relapse and to exhibit a periodicity in the relapses. Remak observed relapses at intervals of three months; Von Hasner, every month; Clark, every week; Moebius had a patient in whom the crises returned during the month of August in three successive years; Thomsen, one in whom they occurred in May and October. In time this periodicity gives place to a permanent condition. The author does not agree with Charcot's opinion that these two successive stages of periodicity and permanence are independent. The phase of periodicity lasts for a variable time with accompanying phenomena of malaise, nausea, vomiting, photophobia; in a word, the symptoms of ordinary migraine. The paralytic period follows this at an interval varying from hours to weeks. There is usually a rapid succession in this order: the upper lip drops, the eye turns outwards, the motor feebleness appears throughout the distribution of the third pair; the patient suffers with diplopia, complains of an inability to read at an ordinary distance due to paralysis of accommodation. This second state lasts from hours to months, the phenomena usually disappearing rather rapidly, but not suddenly, to return in the order in which they previously occurred after an interval more or less considerable. The pathological anatomy remains a matter of hypothesis. The suggestion of Charcot was that the disorder was wholly functional, but that by repetition these troubles determined organic alterations at the region of the oculomotor nerves. The treatment suggested is a long course of bromide with the addition of iodide of potash for the circulatory disorders, and electricity in case the paralytic symptoms are persistent.

MITCHELL.

MIGRAINE ALTERNATED WITH METATARSALGIA.

At the French Congress of Alienists and Neurologists, M. Lamacq (La Médecine Moderne, Aug. 12th, 1896) described a single curious case in which the patient had a regular alternation of neuralgia of the right foot with right hemicrania. The trouble of the foot is not minutely described in the report, but L. calls it Morton's disease. The duration of the two phenomena was identical, either lasting about twenty-four hours. If the course of the foot-neuralgia was arrested, as by a warm bath, it was replaced in a few hours by migraine. During the attack of metatarsalgia the same general accessory symptoms were present that would accompany a classical migraine: pressure in the head, difficulty in intellectual application, loss of appetite, nausea, etc. A perfect equivalence seems to be made out for the two phenomena.

MITCHELL.

TWO CASES OF MORTON'S DISEASE.

At the French Congress of Alienists and Neurologists, M. Lamacq (La Médecine Moderne, Aug. 12th, 1896) describes two cases which he calls Morton's disease, the first of which was rapidly cured by sulphur baths; the second, in which neurasthenic symptoms played a part, disappearing more gradually. There was no relaxation of the plantar arch in either case, and both of them seemed rather to have been ordinary cases of neuralgia, possibly rheumatic in origin, than to conform to the classical type of Morton's disorder.

MITCHELL.

CONCUSSION OF THE SPINAL CORD. Willard and Spiller. N. Y. Med. Jour., March 6th, 1897.

These authors give us the results of a case of fracture of the eleventh thoracic vertebra. The patient received a severe blow from

a trolley car in this region. The lower limbs were completely paralyzed, no involuntary movements were noticed. Sensation lost entirely below Poupart's ligament, except on the front and outer parts of the thighs (ext. cut. nerves). Inability to urinate and defecate was presented. The 11th thoracic vertebra was found elevated, the 12th depressed. Death from exhaustion. The autopsy revealed considerable extravasation of blood into the muscles and connective tissue at the seat of injury, the laminae of the 11th thoracic vertebra were fractured, and there was extradural hemorrhage. No displacement of the vertebral bodies. The dura was intact, and no hemorrhage was observed within it. The cord was firm, of normal shape, and showed no signs of softening exteriorly. No indications whatever were evident macroscopically of pressure on the cord, microscopically were found displacement of fibres in one portion of the cord, numerous hemorrhages, altered blood pigment, masses of granular corpuscles, necrosed tissue, swollen axis cylinders, tumified ganglion cells and round cell infiltration. The spinal roots contained a few swollen axis cylinders, and the medullary sheaths did not stain quite so deeply with haematoxylin as normally. Blood vessels everywhere much dilated. The pathological results are the more important as they occurred early, the patient dying five days after injury. After citing various cases bearing upon their subject, and one recently published by Westphal similar to their own, the authors call attention to the facts recorded in this case. Paralysis of the lower limbs ensued immediately after injury, yet no lesions due to compression through fracture pressure were found. They consider the symptoms due to the pathological changes found in the cord, and the case one of so-called concussion of the spinal cord in which condition we are not wont to await clear structural changes. The communication is of great importance, and contributes not a little to the possible pathology of "railway spine" and the traumatic neuroses. When we consider that cases of this character, unless there be serious damage to the cord which can be determined exteriorly, seldom come to autopsy early after the injury, the changes enumerated above assume more dignity. Their significance for those cases which, after a lapse of time, we usually consider purely functional in nature, *i. e.*, traumatic neuroses, traumatic neurasthenia, etc., should not be underestimated especially as these cases so frequently demand recognition before courts of law.

STERNE (Indianapolis).

TRAUMATIC NEUROSES. A. L. Hall, M.D. (Med. Record, Sept. 26th, 1896).

The surgeon should be an equal authority with the neurologist in determining the sequences of trauma upon the nervous system. Neurasthenia is the usual form under which traumatic neurosis expresses itself, and its symptoms are indistinguishable from neurasthenia arising from other causes. The actual condition of the patient previous to the accident must be known in order to reach a correct estimate of the injury sustained by the nervous system. The type of symptoms manifested, whether neurasthenical or hysterical, is often a question of vital importance in the adjudication of a claim for damages. Traumatic neurosis occurs oftenest at the centres of population, but is by no means rare in country districts. It is probable that traumatic neurosis is dependent upon some definite, yet unknown change in the arrangement and structure of the cellular elements of the nervous system, which gives rise to stable rather than unstable symptoms. A stable, well-organized symptom-complex indicates damage to the nervous structures, while instability of symptoms and want of orderly arrangement denotes trivial injury, and if long continued, simulation is rendered probable. The so-called "objective symptoms" depend upon the psychical rather than the physical state of the subject, and

are unreliable guides to diagnosis. A correct diagnosis is best obtained from a reliable account of the accident, the previous history, the presence of surgical troubles, and the existence of a stable, well-defined, organized symptom complex. The term, traumatic neurosis, is an expression for an indefinite condition, and a simplification of the subject is desirable from a clinical standpoint. FREEMAN.

TETANY. Boston Medical and Surgical Journal, Nov. 19th, 1896.

Howard A. Lothrop, M.D., reports three cases, one of which was in an adult, a pregnant woman, thirty years of age, native of Syria. The salient points of this case were recurrent attacks of tonic spasm of symmetrical groups of muscles in the arms and legs, preceded and accompanied by pain, numbness and prickling. At the height of the spasm the patient was powerless to move either fingers or toes, and was utterly unable to walk. Fibrillary contractions of the facial muscles were marked and constant about the orbits. Gentle percussion over the larger branches of the facial nerve on either side provoked violent contractions in the corresponding muscles (Chvostek's phenomenon). The arms were held at the side and flexed at the elbow; the hands were in the position known as *main d'accoucheur*. There was flexion at the wrist and metacarpo-phalangeal articulations while the two distal phalanges were extended. The thumb was drawn across the palm of the hand, which was made hollow by the over-prominence of the thenar and hypothenar eminences. Both hands were symmetrical and the position was maintained by tonic muscular spasm. The flexor muscles of the forearm and the smaller muscles of the hand were very tender and painful as a result of this forced muscular action. Pressure on the ulnar nerve above the internal condyle served to increase the pain and render the muscles more tense. Voluntary motion was impossible, and her hands were useless during the spasms, which lasted from a few moments to an hour or more. The tendon reflexes were absent during an attack of spasm and were normal in the interim. Attacks of spasms in the hands and feet could be obtained at will by nerve pressure as first described by Trousseau. There was increased excitability of the nerves to electric currents, particularly the galvanic, as demonstrated by Erb and Hoffman. These symptoms developed about the seventh month of pregnancy and diminished as she approached the time of her confinement. The labor was perfectly normal and there have been no further spasms since the birth of her child.

The other cases occurred respectively in a boy aged three and a rachitic male child eighteen months old. SHIVELY.

TETANY IN CHILDHOOD.

Kalisher—Yahrbuch für Kinderheilk., Band xlii., Heft 3 und 4—discusses the frequency and characters of tetany in childhood. As diagnostic criterion he gives the presence of tonic spasm without loss of consciousness, affecting mainly the interossei and small hand muscles, producing flexion of the proximal and extension of the middle and end phalanges with strong adduction of the thumb and pressing together of the fingers—"obstetric hand"—the foot generally in equinovarus position, the great toe overextended the other toes flexed. The symptoms of Chvostek and that of Trousseau are generally but not always present. They occur also in other diseases. Increased electrical irritability is more constant. An elaborate scheme for examination of cases is given. In a study of a material of 2191 cases under 2 years K. found 1141 cases of rickets, with 3 cases of tetany, while among the 1077 cases free from rickets there were 2 cases of tetany, a total of 5 cases among 2191 patients. He concludes that while rickets may be a predisposing cause, it can hardly be considered to have a very close relation to tetany. An examination of the literature of the subject and his own experience convinces him that tetany is a

rare disease, though somewhat more common in young children than in adults. He regards it as similar in etiology to the other spasmodic disorders, and finds it, in children, quite amenable to treatment.

C. A. ALLEN.

TETANY FOLLOWING SCARLATINA.

Bradford McConnell (Canada Med. Record, Sept., 1896) reports a typical case of tetany following scarlet fever. The inception of the trouble was noticed fourteen days after the beginning of the fever and during desquamation. The specific action of the infectious disease in causing the spasmodic affection is more than doubtful, as the patient, aged 5½ years, had had a similar attack at 18 months. There was a vicious heredity, and the child himself had had numerous convulsions.

PATRICK (Chicago).

EIN FALL VON TETANY MIT EIGENTÜMLICHEM SECTIONSBEFUND. (A Case of Tetany with Peculiar Findings.) Deutsche Zeitschrift für Nervenheilkunde, Band ix., Heft 3 und 4. By H. KÖSTER.

The diagnosis of tetany in this case was founded on tonic, intermitting frequently painful cramps of individual muscles or groups of muscles, on the presence of Trousseau's and Erb's signs, on intermitting spasm of the ocular muscles, retention of urine, dysphagia, tinnitus aurium, absence of mental symptoms, vertigo and the development of the disease in the first months of the year.

The simultaneous appearance of the cramps in all four extremities, affecting the extensors as well as the flexors, the flexion of the leg, the intensity of the spasm in the neuchal muscles, the ability of the patient by an effort of the will to control the cramps, the position of the hand as a fist, the clonic convulsions, at times, in the muscles of the extremities and eyes, were peculiarities noticed in this case. A slight rigidity of the muscles prevented the knee jerk. The autopsy revealed contracted kidneys, circumscribed hemorrhages on the anterior part of the cauda equina and about the roots of the third to the fifth cervical nerves, as well as hemorrhages and hyperæmia in the sheath of the right sciatic nerve.

Köster is unable to explain the symptoms from the post-mortem findings.

SPILLER.

PATHOLOGICAL ANATOMY.

ASCENDING DEGENERATION IN NERVES AND THE CONSEQUENT CHANGES IN NERVE CELLS.

Fleming has made an unusually extensive and careful study of the changes that occur in the central stump, and in the cells of the spinal cord after division or ligation of the nerve trunk.

In the nerve he found: "First, that in process of time a slow atrophy of 'motor' fibres occurs. Secondly, that certain 'sensory' fibres degenerate centrally, possibly because severed from their peripheral trophic centres. Thirdly, that the minute fibres found in a normal nerve undergo very marked change. Fourthly, that distinctive connective tissue increase occurs."

The changes found in the cells are summarized as follows:

"1. The cells of the ganglia on the posterior nerve roots undergo definite changes as the result of nerve section or ligature, and do so at a much earlier period than the multipolar cells in the cord—beginning probably as early as the fourth day and certainly by the seventh day.

"2. That one of the very first changes observed in the cells of ganglia and anterior cornu is a diminution in the size of the nucleus—in proportion to the size of the cell and that, sometimes, but not in all cases—nucleoli also becomes smaller and very frequently the nuclei take up an eccentric position, sometimes even bulging the cell wall.

"3. That in both sets of cells Nissl's granules, otherwise known as the chromatic granules, are either smaller in size, fewer in number and scattered through the cell body tending to be most numerous round the nucleus, or else they are grouped together in large masses round the nucleus, leaving the periphery of the cell quite clear.

"4. That pericellular lymph spaces may become enlarged, especially in the ganglia cells, and where the enlargement is very marked, the cells become proportionately smaller in size—although an actual atrophy may also occur. In several of my specimens I found large vacuoles—not the vacuoles described by many writers as occurring in the cells of the cord and cerebral cortex, which are probably to some extent artificial—but big vacuoles more resembling distended pericellular lymph spaces. They differ, however, inasmuch as they are surrounded by the remains of cell protoplasm containing chromatic granules.

"5. That in the multipolar cells not merely are there these changes in position and size of nuclei, and arrangements and number of chromatic granules, but there is as a later phenomenon, marked disintegration of cell protoplasm, well seen in some of my specimens. This disintegration has been described by Marinesco as occurring in certain cord lesions in man. It consists of patches—which with toluidin blue and eosin, are whitish in color and surrounded by masses of chromatic granules."

PATRICK.

THE ALTERATIONS OF THE NERVE ELEMENTS IN EXPERIMENTAL UREMIC DYSCRASIA.

Drs. Sacerdotti and D. Ottolenghi, of Torino, have made very interesting experiments on the effect of uremic poisoning upon the various nerve systems, and have published their results in the January, 1897, No. of the *Rivista di Patologia Nervosa e Mentale*.

Their experiments were made on four dogs and three rabbits. In three dogs the kidneys were extirpated at one time; in the fourth 75 days intervened before extirpation. In one rabbit the kidneys were removed, and in the other two the ureters were ligated near their entrance into the bladder. Of the dogs, one lived four days, 2 hours; the second, 5 days, 9 hours; the third, 7 days, 10 hours; the fourth, 4 days, 10 hours. The rabbit whose kidneys were removed lived 50 hours, the others 48 and 56 hours respectively.

The macroscopic examination of the brains of these animals offered nothing of importance. A certain amount of hyperæmia was present in two of the dogs' brains, while in the other two the brains were anæmic. The brain of the dog that lived 7 days showed a slight diffuse opacity of the pia, and a slight amount of softening of the superficial strata of the cortex. The rabbits' brains showed nothing appreciable. The methods employed were those of Golgi and Nissl.

The ganglion cells of the brain cortex, of the hippocampus, cerebellum, and the neuroglia cells showed a degeneration or rather a varicose atrophy of the protoplasmic prolongations in various degrees.

The authors draw the following conclusions from their experiments:—

1. That ligation of the ureters or bilateral nephrectomy provokes in the nerve centres lesions easily demonstrated by Golgi's method. Lesions characterized by a varicose atrophy of the dendrites of the ganglion cells, while the axis cylinder prolongation remains unaltered. Also that, contrary to the teachings of Acquisto and Pusaterie, the neuroglia cells do participate, showing varicose degeneration of the prolongations of the cells.

2. That regarding the distribution of these lesions—

- a. The elements having undergone degeneration are most diffuse in the whole cerebral cortex and appertain to the different layers of cells.

b. Less numerous than in the cerebral cortex, but yet abundant, are the altered cells in the foot of the hippocampus;

c. In the cerebellum the degenerated cells are found in the molecular stratum;

d. The neuroglia in the whole region studied is more or less altered.

KRAUSS.

THE NEUROGLIA, NORMAL AND PATHOLOGICAL.

F. W. Enrich has published the results of some careful work with the new Weigert stain for neuroglia (*Brit. Med. Jour.*, Oct. 10th, 1896).

"The results obtained by this new method are somewhat at variance with the views held by Golgi and others. The latter believed that the neuroglia consists of an intricate feltwork of cells and their branches; Weigert is of opinion that in the adult, at least, these fibres or branches become differentiated from the protoplasm of the cell body. The usefulness of a method increases in proportion as it is employed side by side with other methods. Golgi's process has demonstrated the epiblastic origin of neuroglia; the shapes and forms of its primitive elements in the fœtus and their relation to nerve cells, nerve fibres and blood vessels; comparative anatomy has shown the evolution of the neuroglia cell; and Weigert's stain shows the ultimate stage of this neuroglia, and its distribution in the human adult. Not all the neuroglia appears to reach this last stage, in which the fibre has become differentiated from the cell protoplasm; some probably remains in the pure cell condition, but ready to form the ultimate fibre when called upon to do so. Whether these newly-formed fibres which constitute all forms of sclerosis and all cicatricial processes within the central nervous system are formed from cells which have rejuvenesced, as it were, after having in the natural course of their development given off the full-formed fibre, or whether the younger type of neuroglia cell is the parent, cannot as yet be determined. Weigert's view as to the nature of adult neuroglia has been objected to on technical grounds; the neuroglia fibres even in the adult are said by these opponents to be still real cell processes; a study of the processes going on in the cerebral cortex in general paralysis sufficiently refutes this. Of the functions of the neuroglia we know little; probably a more complete study of the distribution of that tissue will aid us. Possibly its function varies with each step in the development of the nervous system; at first it directs the growth; then it supports fibre and cell; and lastly, it insulates. In addition it probably acts as a fourth coat to the blood vessels, which, in the central nervous system, are notoriously in adventitia. Abnormalities in the formation of the neuroglia may possibly affect detrimentally these functions; but this subject requires a further and vaster amount of study. Neuroglia cells have also been supposed to have a "scavenger" function in some morbid processes, and to be part of a lymph connective system; this theory, however, is very improbable in the face of the development and distribution of the neuroglia elements. Some interest attaches to the relation between syringomyelia and so-called central "gliosis"; Weigert appears to be of opinion that the former cannot result from a softening of the latter. Another interesting feature is the varying direction of fibres in secondary sclerosis. The majority run parallel to the course of the degenerated axis cylinder; but some, for example, in the spinal cord, run at right angles to it, and outwards beyond the area of a nerve degeneration, till they reach the peripheral zone of neuroglia which becomes thickened. This secondary hypertrophy of the subpial layers has perhaps now and again been mistaken for a peripheral degeneration of nerve fibres."

PATRICK (Chicago).

DEGENERATION FOLLOWING LESIONS OF THE OCCIPITAL LOBE.

Shaw (*Brit. Med. Jour.*, Sept. 12th, 1896) reports the results of careful study of the degenerations caused by lesions of the occipital

lobe in ten monkeys. In some the entire occipital lobe was removed, in others a circumscribed lesion was made. The following summary is a very brief statement of the findings and conclusions of the author.

"1. A lesion, therefore, of the cortex of the occipital lobe gives rise to descending degenerations.

"2. The degenerated fibres pass down by way of the corona radiata.

"3. Having reached the level of the corpus callosum, a division into the main groups occurs; one group transverse this commissure to reach the opposite side.

"4. Of those which remain on the same side of the lesion, the majority occupy the hindermost part of the capsule, and end in the grey matter of the pulvinar, external, geniculate body, and anterior corpus quadrigeminum. Others are found scattered throughout the posterior two-thirds of the internal capsule, and descend into the crusta of the crus cerebri.

"5. Of the crossed fibres, most pass into the white matter of the opposite occipital lobe, and no doubt end in the cells of its cortical grey matter. Others, however, turn forwards and descend in the internal capsule, occupying its posterior two-thirds. These ultimately reach the crusta of the crus cerebri.

"6. Some of the degenerated fibres occupying both crustæ probably end in the gray matter of the substantia nigra, while others reach the pons.

"7. Degenerated fibres also pass towards the posterior commissure; some of these traverse it to gain the opposite side, and turn downwards; others remain uncrossed. Both sets apparently end in the grey matter around the aqueduct of Sylvius. These supply a connection between the cortex of one side and the nuclei of nerves to the eye muscles on both sides.

"8. A few degenerated fibres were found in the posterior longitudinal bundles on both sides, probably some of the set last described. Others, inconstant in number and situation, were found in most of the medulla and cord. No importance was attached to these latter."

PATRICK (Chicago).

THE PATHOLOGY OF MULTIPLE SCLEROSIS. By Prof. A. Strümpell (Neurologisches Centralblatt, No. 21, 1896).

Strümpell has been unable to confirm the statements, made by Marie, that multiple sclerosis in most cases follows infectious diseases. In at least thirty or forty patients examined by the writer, it was exceptional to find any connection with infectious diseases. Neither can he agree with Oppenheim in regard to the influence of toxic agents (lead, alcohol, etc.) in the production of the disease.

It is not probable that the morbid process has its origin in the vascular system. If this were true, it would have to be a vascular disease which begins exclusively in the smallest vessels of the central nervous system, as there are no corresponding alterations in the other organs of the body.

Strümpell is inclined to regard multiple sclerosis as an endogenic process; as one which has its origin in congenital malformation. He has observed a case of hydromyelia combined with central gliosis and disseminated sclerosis, which he explains in this way. The frequent commencement of the disease in youth, and the preservation of the axis cylinders in the sclerotic areas for a long period are in favor of this opinion. In almost all exogenic processes the nerve cells or axis cylinders are early affected. It is probable that in multiple sclerosis the disease begins in the proliferation of the neuroglia.

Strümpell states that in most cases of disseminated sclerosis the intention tremor in no way differs from the ataxia seen in tabes, Friedreich's disease, etc. As ataxia of an intense degree may be noticed

in multiple sclerosis without the slightest trace of sensory disturbance, the latter can not be regarded as necessary for the manifestation of ataxia.

The writer also calls attention to the fact that the abdominal reflex is often absent in multiple sclerosis.

SPILLER

INFANTILE AND HEREDITARY MULTIPLE SCLEROSIS. By Prof. H. Eichhorst (*Virchow's Archiv*, 146-2, 1896, p. 173).

Although Marie, Unger, Moncorvo and Nolda have reported cases of multiple sclerosis occurring in children, there has not been as yet a single autopsy confirmative of the opinion of these writers.

One of the cases reported by Eichhorst in this paper is the first which offers a foundation for their statements, and both are interesting in another way, inasmuch as they prove unquestionably (Eichhorst) the possibility of hereditary transmission of the disease.

A woman, who had multiple sclerosis, gave birth to a child who developed symptoms of the same disease. The first distinct manifestations of the malady were noticed in the boy at the age of eight, but his father stated that he had never been normal, and had had a tremor for a long time, which was increased by voluntary movement. Death occurred when the child was nine years old. Three other children in the family were healthy.

Autopsies were obtained in both cases, and disseminated sclerotic areas were observed, which did not extend beyond the spinal cord, and yet cerebral symptoms of a severe type had been observed in both mother and son. According to Eichhorst, the clinical manifestations frequently do not agree with the post-mortem findings in disseminated sclerosis. The symptom-complex seen in this disease may be the result of a functional condition. In both cases the sclerotic foci were very small.

SPILLER.

PATHOLOGICAL CHANGES IN AMYOTROPHIC LATERAL SCLEROSIS.

In a report of two cases of amyotrophic lateral sclerosis in the *American Journal of the Medical Sciences* for June, 1896, Dr. Joseph Collins makes an interesting contribution to the pathology of the disease. In his first case the pathological findings are summarized as follows:

"In cervical portion of cord in fresh state, from third to sixth segments reddish, softened appearance in the area of anterior horns. Almost complete degeneration of crossed pyramidal tracts, from the medulla to the end of the cord. Degeneration in the uncrossed pyramidal tracts, but not so extensive or complete. Evidence of degenerated blood vessels and excessive vascularization throughout the cord, especially in the cervical and dorsal regions and most evident in the grey matter. Massive atrophy in the ganglionic cells of the anterior horns throughout the cord, remarkably so in the cervical region, where they are almost entirely absent, and the few that are to be seen in a section are in a high state of degeneration. Increase of the spider cells, particularly in the cervical region. Best preservation of cells of any individual column are the cells of the groups of the lateral horns. Central canal filled up and distended with a proliferation of the ependyma. Capillary hemorrhages in the cervical grey matter, associated with rarefaction of the ground-substance of the anterior horns, and a condition of necrobiosis. Striking degeneration of the twelfth nerve nucleus throughout its entire extent, except at its very termination ventrad. Slight degeneration in the common accessorio-vago-glossopharyngeal nucleus of the tenth nerve. Nucleus of the seventh nerve apparently normal. No changes in the motorial pathway above the pons, and cortical cells of the motorial areas entirely normal. Atrophy of the trunks of the twelfth, the ulnar, etc., and degeneration to be seen microscopically. Usual changes in the muscle fibres"

In his second case there was:

"1. A marked sclerosis of the crossed pyramidal tracts, moderate degeneration of the direct. 2. Marked atrophy of the cells of the anterior horns of the spinal cord. 3. Markedly dilated and thickened blood vessels, rather diffusely distributed. 4. Atrophic changes in the cells of the twelfth nerve nucleus. 5. Destructive myelitis of the cervical region, tuberculous in character. 6. Hemorrhage, of ancient date, in the dorsal region."

H. L. S.

PSYCHIATRY.

RIGHT ZOSTER AND GENERAL PARALYSIS.

At the Soc. Med. des Hôp., Nov. 27th, 1896 (Medical Week, Dec. 4th, 1896), Dr. Danlos reported having under his care a patient suffering from right zoster ophthalmicus, with no perceptible disturbance of the nervous system, who was admitted six months later at the hospital with distinct symptoms of progressive general paralysis. Dr. Danlos raised the question whether this was merely a coincidence, or whether there was a connection between the zoster from which the patient had suffered, and the paralytic condition. Dr. P. Marie thought it difficult to determine whether the zoster had preceded or followed the development of paralysis, and he considered it quite possible that the first symptoms of the latter affection had escaped observation. One is often struck with the marked nervous affections, particularly in the region of the sympathetic, associated with zoster. Dr. Rendu added that he thought zoster invariably an expression of severe disturbance of the nervous system, and that in one of his own cases an apoplectic attack had been followed by zoster in one of the paralyzed limbs.

MITCHELL.

DEMENTIA PARALYTICA. Tod durch Suffocation. Etat criblé. Cystöse Degeneration. Meningitis Tuberculosa. [Dementia Paralytica, Death by Suffocation. Etat criblé, Cystic Degeneration, Meningitis Tuberculosa.] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. 146, Heft 3. By Robert Neudörffer.

Small cysts found within the brain substance in a case of dementia paralytica were supposed to have been caused by the cysticercus, although doubt was entertained on account of a small vessel found within one of these cysts, which had been cut open, and of the absence of an embryo and hooklets. More careful examination of the brain disclosed numerous small cavities, in many of which a small vessel could be found. It was evidently the condition described by Durand-Fardel as état criblé. The cystic degeneration of Rippling, according to Neudörffer, is in reality the same process. The formation of these cysts is probably the result of enlargement of the perivascular lymph spaces. Durand-Fardel attributed them to chronic congestion, but it is probable that interference with the flow of the lymph plays an important rôle. Etat criblé is observed in senility, alcoholism and progressive paralysis. In all of these processes congestion occurs.

Tubercular meningitis on the convexity of the brain was an unexpected discovery at the autopsy. It is a well-known fact that certain diseases, especially phthisis, influence the course of psychoses, and vice versa. In this case the tubercular meningitis was probably the cause of the somnolent, apathetic condition. Heubner has stated that tubercular meningitis may present the picture of a psychosis. The patient died from suffocation while eating. Death was probably due to some disturbance of deglutition, possibly caused indirectly by the meningitis.

SPILLER.

INFECTIOUS DISEASES IN GENERAL PARALYSIS.

Delmas (Arch. Clin. de Bordeaux) from a study of the literature and his own experience is of the opinion that parietic dementia is not rarely caused by infectious diseases, although it is almost impossible to prove it. Indeed, the evidence adduced seems to us to be singularly insufficient. One of the cases is simply one of rather marked delirium and great prostration during puerperal infection, the patient being fully recovered at the end of four weeks. The author's conclusions follow.

It is generally admitted that general paralysis, in a great number of instances, is due to syphilis which acts by virtue of being an infectious disease and by means of its toxins.

Facts show that acute infectious disease may be quickly followed, in some cases, by typical dementia paralytica, and even more frequently by alienation (acute delirium, confusional insanity, pseudo-parietic dementia) which presents more or less completely the clinical and pathologico-anatomical characters of parietic dementia.

Infection, then, would seem to play an important rôle in the production of general paralysis of the insane and similar affections, and it is pertinent to inquire if general paralysis and psychopathies approximating it are not prominently diseases of infectious origin.

PATRICK (Chicago).

LONG DURATION OF GENERAL PARALYSIS.

M. Lapointe (La Medecine Moderne, Aug. 5th, 1896), at the French Congress of Alienists and Neurologists, reported briefly a case of general paralysis which gave rise to a long discussion and the report of several interesting cases. M. L. had observed a case of general paralysis of unusual duration in which the cardinal symptoms had gradually disappeared and been replaced by simple dementia. The autopsy verified the diagnosis after the disease had lasted fifteen years.

M. Vallon had seen similar cases verified by autopsies and considered that the maniacal form had frequent remissions but the dementia type had none. He found it had important prognostic sign when a general paralytic was losing flesh, and believed that this indicated a rapid course of the malady; on the other hand, if there was an increase in weight he thought it likely that the patient would live a long time.

Voison and Séglas had observed similar cases, lasting fifteen to twenty years, with marked symptoms during the whole of that time of general paralysis.

Doutrebente and Régis thought that the diagnosis in such cases was very likely to be confused with that of disseminated sclerosis, with cerebral syphilis or with chronic alcoholism.

MITCHELL.

GENERAL PARALYSIS OF THE INSANE AND ITS TREATMENT.

(Discussion before the British Medical Association. Brit. Med. Jour., Sept. 26th, 1896.)

Macleod said that in treatment seclusion was absolutely necessary and no sedative of any benefit. In the last stage all that was required was attention to the bladder and bowels and ordinary nursing.

Rayner thought over-feeding and narcotics were often very harmful.

Clouston said that in the early stage he was now in the habit in specially acute and risky cases of combining seclusion with sulphonal. He gave the sulphonal in from 30 to 40 grain doses, dissolved in hot milk, the method of administration which a German experimentalist had found entirely to prevent hematoporphyrinuria. After the first day the patient became sulphonal drunk. About the third day a condition little short of sulphonal coma was produced, and then he diminished the dose. After a week or ten days of this treatment the general paralytic passed quietly and without accident into the second

stage of the disease. He had tried this plan in several cases, and, looking to the incurability of general paralysis, he believed that it was one that they were justified on medical grounds in adopting in a certain number of carefully selected instances.

Mickle, in the early stage of excitement, relied principally on diet, purgation, baths, cold to the head and seclusion.

Turnbull said that for cases in the third stage he used a mattress in three sections, in the centre of which there was placed a small water cushion and a tube for training off the urine. Turning of the patient was thus rendered unnecessary. He had found that bed sores which would not heal when the patient was on an ordinary mattress often closed when an apparatus of this kind was resorted to.

Seymour Tuke had also noticed a change of type in general paralysis. Whether education had to be considered as a point in this change—and especially in the change of type of delusion—had to be further considered. Dr. Clouston's remarks on the sulphonal treatment were exceedingly interesting, but the cases would have to be carefully selected, to say the least.

Campbell Clark said that Dr. Clouston's plan of treating early cases was an attempt to push the patient through the first stage as quickly as possible, and he thought this involved a certain amount of risk. He was not sure that this treatment would always be so successful as Dr. Clouston had found it. It would be of great importance to be able to give a definite prognosis as to whether the disease was going to run a short or a long course. He had not been able to confirm Beavan Lewis' views on this subject especially with regard to the pupil symptoms to which he had attached so much importance in the matter of prognosis.

Yellowless expressed his distinct disapproval of Dr. Clouston's method of treating the first stage.

Dr. Clouston thought that he must have expressed his views as to the sulphonal treatment so as to be misunderstood. As a matter of fact he had only used it in that way in about three cases within the last five years. They were extremely bad cases.

Regarding the frequency of the disease, Drs. Clouston, McDowall and Turnbull believed it to be very decidedly on the increase. Regarding etiology, Dr. Clouston said that he did not believe in the syphilitic origin of the disease, though he admitted that there were facts which might point that way. Campbell Clark had concluded that only a very small proportion of the cases could be attributed to syphilis.

PATRICK (Chicago).

STUDY OF THE BLOOD IN GENERAL PARESIS. By Jos. A. Capp (Am. Journal of Medical Sciences, June, 1896).

One of the most notable and worthy studies of the year in the United States is the one above, notable because one of so few of like character; worthy because a result of painstaking labor in a line promising small practical immediate results. This work is really an outcome of the McLean Laboratory. It is systematic in that it begins with a review of all previous findings. These previous studies are not numerous, and were not made to be inclusive of all points. Notably among them, McPhail found hæmoglobin low and white corpuscles increased in the later stages of general paresis; Lewis found about the same; D'Abundo, that blood from a paralytic is more toxic than from a normal person. Other observers have reported on very few cases and with no specially novel findings.

The author details his method as showing the efforts to secure accuracy, and gives the accuracy of cells used (Ehrlich's). The time selected for the examination was from three to five P.M. and during digestinal leucocytosis. Ten normal adults in health were first examined under similar conditions for control tests, these not showing any very marked increase as due to digestion.

He finds from examination of nineteen cases that (1) the hæmoglobin and red corpuscles are always diminished; (2) most cases show a slight leucocytosis amounting on an average to about 22° above the normal—early cases may have no leucocytosis whatever; (3) in the differential count a decrease is found in the lymphocytes along with a marked increase in the large mononuclear cells. The eosinophiles in a few cases are very numerous.

Convulsions and apoplectic attacks:—1. The red corpuscles and hæmoglobin are usually increased at the time of a convulsion. During an apoplectic attack of long duration they are both somewhat diminished.

2. The specific gravity is variable, sometimes increasing, sometimes diminishing, at the time of an attack.

3. There is a leucocytosis after convulsions and apoplectic attacks, which is as sudden as it is usually pronounced. It certainly does not appear within a very short time preceding the convulsion, probably not before it actually takes place.

4. The degree of leucocytosis and the period of its continuance, as a rule, vary directly with the length and severity of the attack.

5. In the production of the leucocytosis the large mononuclear cells are increased relatively more than any other variety.

6. The fact that after convulsions and apoplectic attacks in general paresis there is not only an increase in the number of white cells, but a change in their character, as shown by the differential count, and at times abnormal cells appear, is an argument against the theory that leucocytosis is merely a change in the distribution of the white corpuscles.

PHELPS.

POLYNEURITIC PSYCHOSES. By Emil Redlich, M.D. (*Wiener klinische Wochenschrift*, Nos. 25, 26 and 27, 1896).

It was Korsakow who first made psychoses due to polyneuritis well known, and showed that they could not be considered merely as the effect of alcohol, inasmuch as they were observed in polyneuritis from other causes. He attributed the mental condition to the presence of toxic substances in the blood, and called it *cerebro-pathia psychica toxæmica*.

Numerous writers have accepted this view. Wagner has stated that most postfibrile psychoses have a connection with polyneuritis.

Redlich reports two cases of polyneuritic psychosis. In the first case the history of chronic alcoholic intoxication is given, but Redlich attributes the polyneuritis in large part to chronic intestinal disturbance. When the nervous system has been impaired by chronic alcoholism it is especially susceptible to autointoxication. The first manifestation of the psychosis were noticed in mental confusion associated with a condition of excitement and anxiety. This excited state soon passed away, and the patient lost all power of orientation. She was unable to recognize the place in which she was, or the persons about her. She was unable to accept new ideas or to recall the events of the past year, and thus presented the symptom known as retro-antegrade amnesia, which is seen after injuries to the head, hysterical attacks, apoplectic insults, etc. There was no very great disturbance of the intellect, and the events which had occurred previous to the past year could be recalled. A symptom often seen in these disturbances of memory was present in this case. The patient, for example, believed she had just returned from a walk, although in reality she had been for weeks in the hospital, or else that she had previously known the persons about her; such deceptions of memory are not uncommon in the polyneuritic psychoses.

Retrograde amnesia may be in part explained by the fact that during the time the events were occurring the memory was already imperfect, but this explanation will not answer for all cases.

Redlich devotes some attention to the nature of memory. The memory, he states, depends on the intensity of the original impression, on its repetition, on the time which has passed since the impression was made, on the condition of the mind and on the age of the patient.

In the polyneuritic psychoses it is probable that the nervous elements, i. e., ganglion cells and nerve fibres, especially the associative fibres, are so altered by imperfect nutrition that they are incapable of normal function. We are well aware that the toxins are able to produce such degenerative processes. Wagner has accepted this view for certain cases, at least, and inasmuch as recovery is possible he compares the process to the periaxillary neuritis of Gombault, in which the axis cylinders are preserved. The proof of this theory has not been given. In the cases of polyneuritic psychosis, as yet examined, the findings have been negative; this, however, may in part be due to faulty methods.

The second case is in evidence of the fact that in some cases of polyneuritic psychosis delirium may occur. These two cases do not represent in full the clinical picture of polyneuritic psychoses. There is a close connection of amentia (Meynert) and acute hallucinatory confusion with polyneuritis.

SPILLER.

RECOGNITION OF MORPHINE MANIACS.

M. Voisin (*La Médecine Moderne*, November 18th, 1896) at the Société de Hypnologie et de Psychologie, answers the question if a criminal can be recognized as a morphine maniac, in the affirmative, and quotes the following signs and symptoms:

First—Hypnotism, usually unavailable as a means of information, and, indeed, prohibited in France.

Second—Myosis, which is constantly present.

Third—Inspection of the feces, which are gray if the use of morphine has been recent.

Fourth—The traces of hypodermic punctures, present in a majority of cases.

Fifth—The analysis of the urine, which will reveal the presence of the drug if the daily amount of morphine taken is as high as as 10 centigrammes.

At the same meeting, *à propos* of a report of a case of morphine-mania, the Secretary of the Society put the following question to the members: "Is the morphine-user of sound mind, whatever may be the causes of his habit, capable of making a will and undertaking legal obligations?"

A long discussion resulted in the making of a very necessary distinction between a morphine-user and a morphine-maniac, and M. Regnault proposed the following conclusion: "The fact of use or abuse of morphine does not necessarily imply that the person who takes morphine by the mouth, or hypodermically, is therefore subject to a mental disorder which would impair his capacity from a legal point of view. It must always be necessary to examine each case as to signs of mental disorder before deciding whether the person who makes testamentary disposition or undertakes other obligations, can be properly declared of sound mind and capable before the law."

This statement of the case was approved by the Society.

MITCHELL.

CONSCIOUSNESS AND BIOLOGICAL EVOLUTION. From *Mind*, July, 1896. By Henry Rutgers Marshall.

According to the theory of "parallelism," consciousness is a correspondent of biologic evolution, that is, it has its equivalent and concomitant in neural reaction. Marshall states his view substantially as follows: Each and all the elementary activities of life have psychic correspondents—are accompanied by some form of mentality, and under certain conditions this mentality takes the form of conscious-

ness. Neural systematization is of fundamental importance in relation to consciousness, which we may look upon as due to coincident psychic systematization. Different neural symptoms, as in different animals, imply different grades of consciousness. The full consciousness is made up of many subordinate elements, most of which will be made up of many subordinate elements, most of which will be unemphatic, and which will appear to form an unanalysable whole, but some of which will be emphatic and will, therefore, stand out as increments, so to speak, to this unanalysable whole. This unanalysable whole is the empirical ego, and the emphatic elements are the elements which fall within the so-called field of attention—such elements as make up our recognized sensations, emotions, thoughts and acts of volition.

CHRISTISON.

THERAPEUTICS.

THREE CASES OF BASEDOW'S DISEASE SURGICALLY TREATED.

Dr. Fricomi (in *Policlinico*, Fas. 8, 1896) reports three cases of Basedow's disease in which he made a partial resection of both lobes of the thyroid gland. As a result the exophthalmus disappeared, cessation of the tachycardia, of the diarrhoea, of the profuse perspirations, pruritus, and other symptoms, increase of strength and weight of the patients, etc. The author believes that the results obtained warrant surgical procedure, when the ordinary medical treatment fails to bring relief.

K.

THYROIDECTOMY FOR RELIEF OF EXOPHTHALMIC GOITRE. *Annales del Circulo Med. Argentino*, Oct. 15th, 1896.

Dr. Atonio Gandolfo reports a case of Basedow's disease in a patient, 33 years old, who had noticed a swelling of the neck (thyroid) for the past eight years.

On entrance to hospital at Buenos Ayres, a large tumor, bilateral, of the thyroid was found, more voluminous on the right side, with exophthalmus, Graefe's symptom, enlargement of the heart with mitral direct murmur, tachycardia (110-130), thrill in the carotids. Memory is poor, is extremely excitable, has flushings but no chills; skin is pale, anæmic; the whole body trembles. Operated on Aug. 2d, 1895. Ten months after the operation his condition was found to be as follows:—The thyroid enlargement has, of course, disappeared; the exophthalmos has diminished, especially on the right side; Graefe's sign no longer exists, while vision is better and clearer. The heart is still increased in volume, its tones are clear and regular during the day, but at night become irregular, sometimes intermittent; no murmurs; pulse is regular, 90-100 pulsations per minute. The sensation of heat, perspiration, the tremor, and irritability have disappeared, and the patient is regarded as greatly improved.

K.

SECTION OF THE CERVICAL SYMPATHETIC FOR EXOPHTHALMIC GOITRE.

Jaboulay in a recent brief paper (*Lyon Médicale*, Feb. 7th, 1897) reaffirms the efficiency of section, or rather resection, of the cervical sympathetic in the treatment of exophthalmic goitre, and reports three new cases.

The first patient who was 30 years of age, and had presented the triad of symptoms for three years, is reported as cured in a few days.

The second was a woman of 64, suffering from a large goitre, tachycardia, tremor, who had, in consequence of osteoporosis, suffered from a fracture of one rib and the neck of the left femur. The first three symptoms were immediately relieved by operation.

The third patient, a woman of 42, had been for three years the subject of exophthalmic goitre without goitre, who was better from the day after the operation and was ultimately considerably improved.

Besides defending the rationality of the operation, the author asserts that in exophthalmic goitre, as well as in ordinary large goitre, it will invariably diminish the size of the gland and remove in a large degree the danger of subsequent extirpation should such interference be afterwards necessary.

PATRICK.

INTRADURAL SECTION OF THE SPINAL NERVES FOR NEURALGIA.

Robert Abbe, M.D., in the Boston Medical and Surgical Journal, Oct. 1st, 1896, reports three cases in which parts of the posterior roots of the brachial plexus were divided or resected at their intradural origin from the cord for severe and persistent neuralgia. A fourth case in which the four lumbar posterior roots and one upper sacral were divided, is also reported from St. George's Hospital, London, operated on by Mr. Bennett. In all three was marked improvement afforded in the relief of pain, and Dr. Abbe's conclusions are epitomized as follows:—

"A comparatively new and interesting field of work is opened by these few cases.

"Thus far, even in weak patients, the operation has been devoid of risk.

"It is sound in theory, and has yielded enough results to show that it may become a meritorious operation.

"It should be resorted to early in cases of ascending neuritis which have heretofore been subjected to successive nerve-stretching and resection, and finally amputation, uniformly without benefit.

"The experimental and practical evidence shows that two additional roots higher up than the apparent origin of pain should be included.

"There ought to be no risk in severing the posterior roots of the third and fourth cervical, as well as those to the brachial plexus, simply because they supply the phrenic, inasmuch as that needs motor supply only, and at best it has the opposite phrenic in reserve."

SHIVELY.

TREATMENT OF REFRACTORY NEURALGIA BY INTRADURAL RESECTION OF THE POSTERIOR ROOTS.

Dr. Chipault exhibited at the Académie de Médecine (Medical Week, Jan. 22d, 1897) a patient who was operated upon for the relief of severe neuralgia in the right upper limb, by intradural resection of the eighth right posterior cervical root, the pain clearly and persistently manifesting itself in the domain of this nerve. The patient was completely cured, and inasmuch as the recovery had been maintained for two years, it may be considered as definite. No functional disturbance, either motor or sensory, followed the operation; hyperæsthesia persisted barely during twenty-four hours in the area supplied by the divided nerve.

MITCHELL.

RESECTION OF THE ULNAR AND MEDIAN NERVES FOR THE RELIEF OF GENERALIZED NEUROMATA.

Dr. Pean showed at the Académie de Médecine (Medical Week, Jan. 22d, 1897) a man, twenty-five years of age, suffering from generalized neuromata, in whom Dr. P. had resected a large part of the median and ulnar nerves. This operation, as usual, was followed by complete paralysis and anæsthesia in the domain of the nerves; but movement and sensation were gradually restored. At the time of the exhibition the patient experienced no difficulty in the use of his arm, except a little in the thumb. This was ten months after the operation and there had been no recurrence. All the neuromata which existed on the forearm and in the supraclavicular region had become atrophied and disappeared after the operation.

MITCHELL.

ANIMAL EXTRACTS IN THE TREATMENT OF MENTAL DISEASE. (Discus-

sion in the British Medical Association. *Brit. Med. Jour.*, Sept. 26th, 1896.)

Alexander Robertson opened the discussion and reported on the use of fresh sheep's brain, cerebrinin (an extract of the cortex), thyroid extract, thymus gland, and testicular liquid. Here and there a rather striking improvement was noted, but on the whole the results were far from encouraging.

Farquharson had given thyroid to 13 cases of insanity, 5 of mania, 7 of melancholia, 1 of slight dementia. Besides the ordinary somatic signs of thyroid administration, there was nearly in every case some notable change in the mental state; sometimes for better, sometimes for worse, sometimes simply a change in character without notable improvement or deterioration. The ultimate benefit was practically *nil*.

McPhail had used thyroid in about 50 cases and had no doubt that in certain cases it was a most valuable therapeutic agent. There was a mental change in all the cases—in some slight, in others temporary, while in a few the change was little short of marvellous. He could go to the length of saying that we were not justified in considering certain cases chronic after a year or eighteen months without giving them the chance of undergoing thyroid treatment. His limited use of cerebrinin and thymus had been without beneficial result.

Bruce had treated three carefully observed cases with cerebrinin, —30-grain doses of dry cortical tabloids per day. No good results were obtained and no pharmacological action was observed. He reported the results of Dr. Easterbrook's treatment of over twenty cases with ovarin and epididymin, and ten cases with suprarenal tabloids. The results were entirely negative, except that one case of adolescent mania treated with suprarenal tabloids terminated suddenly. That the treatment had anything to do with this is very doubtful, as the same treatment in two similar cases was entirely without result. He himself had no doubt whatever of the efficacy of thyroid treatment in certain cases of insanity. He instanced the case of a patient who had been ill more than two years, and who was regarded as demented. Within five days after the commencement of treatment she showed symptoms of improvement, and within fourteen days after treatment was discontinued, she was practically recovered, and was discharged in three months. He regarded thyroid as of great value in cases of anergic stupor, as a diagnostic between cases of stupor and dementia, and as a diagnostic of curability, as he had never seen any case recover which had not shown some improvement under thyroid treatment. In climacteric, lactational and puerperal cases, where there was a tendency to chronicity, the drug was also of value.

Clouston had used thyroid for two years, and had found the therapeutic effect to be very good in a certain number of cases. In a few it seemed to work a miracle. He mentioned the case of a lady who suffered from adolescent mania, and who after a time sank into what appeared to be confirmed typical secondary dementia. She remained in this state of mental dissolution for over two years, when she was especially sent to the asylum to undergo thyroid treatment. She was first placed under ordinary hygienic treatment, but did not improve. She was then given thyroid treatment, and within ten days after it had begun, she began to waken up in her mind, and her habits improved. She became intelligent, her memory improved, social instincts returned, and within two months she was perfectly well, not only in his judgment, but in that of her family, doctor and relatives. For six months now she had remained well. After such a case, and considering that he had had similar results in other cases, he defied any man to say that the thyroid treatment was of no use in certain forms of insanity. The man who neglected this treatment in such cases could not be said to give his patients the best chance they were entitled to.

Urquhart related a somewhat similar case, Carlyle Johnston had tried thyroid treatment in eight cases, and his results had been entirely negative. PATRICK (Chicago).

SERUM IN MANIA.

Prof. Mariet and Mr. Vires (Medical Week, Sept. 4th, 1896) reported to the French Congress of Internal Medicine a very extraordinary piece of treatment. They injected serum, how prepared is not mentioned, from a "maniac" who had recovered, into two female patients suffering from severe mania. In the first case each injection was followed by marked drowsiness, but no further effect. In the second patient twenty injections were given of 5 c. c. each, followed on each occasion by phenomena simulating drunkenness and deep sleep. The condition improved after each injection, but the excitement returned later. A second series of injections of 20 c. c. in twenty-four hours was given. The improvement which followed was permanent, and the patient completely recovered. The matter is so briefly reported it is impossible to judge whether the results were due simply to improved nutrition, but how this should have been brought about seems obscure. The reporters believe that the hypnogenic properties of the serum are worth considering. MITCHELL.

INSOMNIA OF NEURASTHENIA.

Monin (Independence Méd., July 1st, 1896, N. Y. Med. Jour., Aug. 8th, 1896) says the following mixture is well borne for a long time:

Paraldehyde.....	38 grains
Fl. ext. piscidia.....	75 grains
Syr. cherry laurel.....	750 grains

One dose to be taken diluted.

PATRICK (Chicago).

TREATMENT OF SPASMODIC TORTICOLLIS.

In the American Journal of the Medical Societies for July, 1896, Dr. Maurice H. Richardson and Dr. George L. Walton publish these additional cases treated by radical operation. In their first case the operation consisted of a long open incision, by which practically every contracted muscle was divided. The posterior branches of the upper four cervical nerves were cut and evulsed. The muscles affected were the trepezius, the complexus, the splenius capitis, the trachelo-mastoid, the obliquus inferior, the omohyoid and the sterno-mastoid. The deep muscles inserted into the upper cervical vertebræ—the levator anguli scapulæ and the scalini—were found slightly contracted, but were not cut. The patient was entirely cured. In the second case, the first operation consisting of excision of an inch of the left spinal accessory nerve, was followed by recurrence of symptoms after temporary improvement. A second operation was done, dividing the posterior muscles and nerves, as in the first case. There was immediate relief, but after some months there was a return of slight spasm in the left sterno-mastoid muscle. A third operation was performed. It was found that the nerve had become restored at points of division at previous operation. The nerve was extensively destroyed and the muscle partially severed. This operation removed the last traces of spasm. In their third case reported, a long incision was made from the occiput toward the scapula parallel with the fibres of the trapezius. The muscles were separated and divided until the posterior branches of the four upper cervical nerves were found, of which all the branches were evulsed, including the great occipital. Most of the muscles were also cut across, the trachelo-mastoid alone remaining intact. This last case was greatly improved but not entirely relieved from spasm by the operation.

In view of a recent discussion of this subject, renewed hope of the

benefits of massage, gymnastics and hypnotism, has been encouraged, and a further trial of these methods in aggravated cases is awaited with interest. Surgical measures should certainly not be instituted until every other rational means of non-operative treatment has been exhausted, unless the case is too well-established and severe to justify delay. It may then be resorted to, but as a rational surgical procedure, based upon a by no means discouraging experience.

H. L. SHIVELY.

MESCALE BUTTON IN HEADACHES.

Richardson (N. Y. Med. Jour., Aug. 8, 1896) reports a case of severe neuralgia successfully treated by mescale button (Anhalonium Lewinii). The trouble began as "repeated attacks of occipital and frontal cephalalgias," but the author further along calls them "neuralgias," and after the pains became general he says he "was careful to make a differential diagnosis between neuralgia and acute rheumatism, the diagnosis of neuralgia being thoroughly and unquestionably established." The patient, a man of fifty years, was given four drops of the tincture every night on retiring and this dose was not increased.

"On the evening of the fourth day the neuralgia had left him, being replaced by a peculiar prickling sensation over the surface of the body, but exaggerated in the fingers and toes and also on the soles of the feet. This sensation lasted but a short time, was unaccompanied by any rise of the temperature of the body or change in the character or rate of the pulse; neither was it especially a source of discomfort to the patient, but quite otherwise. After one week this sensation was experienced for an hour or two after taking the drug, but soon ceased and disappeared altogether in the third week. At this time he suffered a very short but severe attack of frontal cephalalgia, which he endured for about eighteen hours, and for exactly two months since he has been totally free from neuralgia. Up to the time of this last attack, this sensation was virtually the only positive action experienced from the drug, but since that time the excretion of urine has been somewhat excessive. Especially would I remark on the clearing of the skin of pimples over the chest and back, and a marked softening of the hair, which before the exhibition of the anhalonium was dry, with a tendency to break easily."

It is proper to state that before mescale button was administered the patient had been subjected to eye treatment and the doctor had treated his stomach expectantly and examined its contents, finding no cause for his trouble there and gave him various prescriptions, which did him no good.

PATRICK (Chicago).

CRANIECTOMY FOR HEADACHE AND MELANCHOLY.

M. A. Voisin (La Médecine Moderne, Oct. 17th, 1896) in an Congress of Alienists and Neurologists described an interesting case of a man twenty-one years of age who had suffered with headache for three years in the left temporo-facial region, which had increased progressively to lypemania, with attempts at suicide. He had convulsions, intractable vomiting, right hemiparesis and facial hemiplegia. After all ordinary treatment had failed craniectomy was performed in the left temporo-parietal region, discovering a cyst four centimetres in diameter in the ascending frontal and parietal convolutions; there was also some pachymeningitis. The various mental and physical symptoms all rapidly improved and the patient was completely cured.

MITCHELL.

Miscellany.

AMERICAN NEUROLOGICAL ASSOCIATION.

The twenty-third annual meeting of the American Neurological Association will be held in connection with the Congress of American Physicians and Surgeons in Washington, D. C., on May 4, 5 and 6. The meeting will be held in St. John's Parish Hall on Sixteenth street. There will be one session daily from 10 A. M. to 1 P. M. During the meeting the following papers will be read:

Report of a Case of Tumor of the Frontal Lobe of the Brain, by Dr. Wm. C. Krauss; Exhibition of a Brain Receptacle, by Dr. Wm. C. Krauss; A Study of a Case of Encephalitis, with Changes in the Pia, by Dr. Alfred Miner; Final Report of the Committee on the After-Care of the Insane, by Dr. Henry R. Stedman; A Case of Tactile Amnesia, by Dr. Charles W. Burr; Two Cases of Acute Ascending Paralysis with Autopsy, by Dr. John Jenks Taylor; Two Cases of Basedow's Disease with Autopsies, by Dr. Charles L. Dana; Five Defective Brains, by Dr. Edward Wyllys Taylor, of Boston; Arachnoid Edema, Productive of Pressure Symptoms after Head Injuries, by Dr. G. L. Walton; Little's Disease, Shall we Retain the Name? by Dr. B. Sachs. Auditory Aphasia, by Dr. Howard T. Pershing; Pathological Anatomy of Huntington's Chorea, by Drs. Joseph Collins and B. Onuf; Microcephalus, by Dr. Frederick Peterson; Anæsthesia in Spinal Diseases, by Dr. P. C. Knapp. 11.30 A.M., Wednesday, May 5: The Nature and Treatment of Torticollis, by Dr. G. L. Walton, discussion by Drs. Dana, Dercum, M. R. Richardson, Gibney and Mills; Steps Toward Insanity, by Dr. Smith Baker; 1. A Case of Syringomyelia; 2. A Case of Acute Softening of the Pons, by Dr. Theodore Diller; Primary Idiopathic Hydrocephalus in Adults (Meningitis Serosa Ventriculorum—Quincke), with a Report of Four Cases, three with Autopsies, by Dr. Morton Prince; A Case of Subacute Bulbar Paralysis, with Exhibitions of Sections; 2. A Case of Syringomyelia, by Dr. Hugh T. Patrick; Report of Committee on Neuronymy, by Dr. Burt G. Wilder; A Case of Purulent Primary Spinal Leptomeningitis, by Dr. Frank R. Fry; A Case of Paralysis Agitans at Thirty-four Years of Age, Immediately Following Typhoid Fever, by Dr. Frank R. Fry; A Report of Two Cases of Tumor of the Brain, by Dr. J. Arthur Booth; Traumatic Focal (Capillary?) Lesion of the Cervico-dorsal Cord, by Dr. F. W. Langdon; Poliomyelitis Anterior Subacuta Unilateralis in a Man of Forty-four—Recovery, by Dr. F. W. Langdon; Some Interesting Cases of Brain Tumor, by Dr. Philip Zenner; Report of a Case of Sarcoma of the Mid-brain in a Child, Associated with Asymmetrical Hydrocephalus, by Dr. C. H. Herter; Epilepsy Following Infantile Cerebral Palsy—Improvement Following Craniectomy and Evacuation of Subcortical Cyst, by Dr. Wm. M. Lyszynsky; Report of a Case of Subcortical Cerebellar Tumor Successfully Removed, by Dr. Joseph Collins and Dr. George E. Brewer; Contribution to the Study of Vertigo, by Dr. Frank K. Hallock; Report of a Case of Jacksonian Epilepsy Relieved by Operation, by Dr. Græme M. Hammond; An

Uncommon Nasal Paræsthetic Neurosis, by Dr. Samuel Ayres; Studies in Scleroderma, by Dr. F. X. Dercum, of Philadelphia; Records of Practice, by Dr. Ralph L. Parsons, of Sing Sing; A Case of Trauma of the Cervical Region of the Spinal Cord Simulating Syringomyelia, by Dr. James Hendrie Lloyd, of Philadelphia.

BOOKS RECEIVED.

Clinical Lessons on Nervous Diseases, by S. Weir Mitchell, M.D., LL.D., Edin., etc. Lea Bros. & Co., Philadelphia and New York.

Telepathy and the Subliminal Self, by R. Osgood Mason, A.M., M.D. Henry Holt & Co., New York.

Syringomyelia, by Guy Hinsdale, A.M., M.D. P. Blakiston Son & Co., Philadelphia.

NOTICE.

The Semi-centennial Meeting of the American Medical Association will be held at Philadelphia, June 1, 2, 3 and 4.

The Missouri State Medical Association will hold its Annual Meeting May 18, 19 and 20.

THE
Journal
OF
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Original Articles.

THE SENSE-AREAS AND ASSOCIATION-CENTRES IN THE BRAIN AS DESCRIBED BY FLECHSIG.¹

By LEWELLYS F. BARKER, M.B.

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ON looking over the field of histology and microscopic anatomy one cannot help being impressed by the fact that the tendency in recent research is towards the solving of problems in two branches more especially of the subject. While the members of one group of investigators are busying themselves with the finer structure of the cell, and of its individual constituents, others are devoting all their energies toward the study of the minute anatomy of the central and peripheral nervous system.

In the domain of neurology during the past decade remarkable advances have been made, advances which are in large part attributable to extension and improvement in our technical procedures. The methods of Golgi and Ehrlich have furnished an abundant store of new facts concerning the complexity of the individual elements of which the nervous system is made up and their relation to one another. The neurones, with their cell-bodies, dend-

¹Read before the Philadelphia Neurological Society, Jan. 25, 1897.

rites, axones, collaterals and telodendria have been placed before our eyes under the microscope with a sharpness and clearness of which no one can have any idea who has not personally interested himself in such studies. Students of forms of life lower than man have garnered a rich harvest of both anatomical and physiological facts which have permitted for the first time the establishment of a comparative neurology worthy of the name, and promise a scientific basis for the development of a comparative psychology.

The study of secondary degenerations has been followed up with extraordinary zeal and the method of Weigert, supplemented by the more delicate procedure of Marchi and Algieri has clearly outlined a large number of the longer tracts in the nervous system which it would have been difficult to localize in any other way. Cytological methods applied to nerve tissues have given us an insight into the interior of the nerve cell, and with the establishment of the existence in it of centrosome and sphere, in addition to the nucleus and the protoplasmic contents the neurone has been brought into line morphologically with the other cells of the body.

The methylene-blue and soap stain of Nissl has enabled us to differentiate in the cytoplasm of the nerve cell two very distinct constituents; (1) a stainable, (2) a non-stainable substance, the former according to Held, standing in direct relation to the food supply of the cell, while the non-stainable substance, on the other hand, represents probably the active living protoplasm, the material basis for the phenomena or irritability.

Not the least important advances, which have been made, we owe to the study of the embryo. The investigations of Balfour, Beard, v. Kupffer, and especially of His, have demonstrated the various modifications which the simple neural tube undergoes before it finally becomes the complex morphological structure which we see in the adult. We have been taught much, too, concerning the histogenetic relations of the individual neurones, the mode of origin of the nerve cells and of the neuroglia, and the various wanderings which many of these cells undergo during the progress of development.

A special developmental problem has been the particular object of study of Paul Flechsig, now Professor of

Psychiatry in the University of Leipzig. It had been noticed in Ludwig's laboratory that the myelin sheaths of the nerve fibres in the central nervous system appear at different periods in different portions of the tissue. With the introduction by Weigert of his hæmatoxylin stain for medullary sheaths, it became possible to study in the central nervous system of foetuses and children the successive steps in the process of the medullation. The "embryological method of Flechsig" has occupied its author for over twenty years, and from it the most gratifying results have been obtained. In 1876 Flechsig published a book entitled "*Die Leitungsbahnen im Gehirn und Rückenmark*," in which he embodied the results of his studies up to that time. He showed that the tracts in the nervous system, which we have good reason to believe are the first to take on functional activity, receive their medullary sheaths also before the others. The myelination of the different bands of white fibres occurs in each embryo in a definite sequence, tract after tract ripening as the physiological capacities of the being become more and more complex. The parts below the corpora quadrigemina including the pons, medulla oblongata and spinal cord become in large part medullated long before the pallium and the interbrain show any myelin at all. The medullation at the time of birth in a full-term foetus, while well advanced in the lower regions of the nervous system, has made but little progress in the higher centres, so that the cerebrum at that time is practically unripe. The human baby at birth is comparable to the dog of Goltz's experiment; it is practically a creature without a cerebrum, although it, like Goltz's dog, is capable of manifold neural activities, some of which are very complex. The experiment of Goltz and the anatomical findings of Flechsig are of the greatest importance, since they furnish us with data dealing with the possibilities of function in mammals of the parts below the great brain when independent of the higher centres. Many complicated movements can be carried out; the evidences of the existence of certain bodily instincts are already present which, if they be not satisfied, lead to muscular movements of a purposeful nature. If the impulses of the newly born be satisfied and if external stimuli of a painful nature be kept away no evidence of consciousness is manifested by the baby; on the other hand, when deprived of

food or exposed to cold, active movements of the body result, often accompanied by loud cries.

Beginning with his Rectoratsrede, entitled "Gehirn und Seele," delivered in Leipzig in 1894, Flechsig has during the past two years on several occasions called attention to the results of his recent studies upon the structure of the cerebrum itself—studies based almost entirely upon his embryological method, although they have been supplemented also by clinical experience and observations of secondary degenerations. I will pass around a copy of the Rectoratsrede and also copies of two important addresses in which the views of Flechsig are outlined, namely, "Die Grenzen geistiger Gesundheit und Krankheit" and "Die Localization der geistigen Vorgänge, insbesondere der Sinnesempfindungen des Menschen."

A full report of all his newer work would be asking too much even of your forbearance. I can only attempt to touch briefly upon the more important features of his anatomical findings and to illustrate by examples some of his main tenets concerning the functional value of different portions of the cerebrum.

Flechsig's work upon the spinal cord and the medulla is doubtless well known to all of you. It still affords better data for judging of the relations of topographical features to function than any other method. I shall pass by, therefore, the question of the myelinization of these parts, and proceed directly to a description of the sequence of the medullation in the parts higher up.

While in the medulla centrifugal conductions with well developed myelin sheaths appear at a time at which the sensory nerves of the medulla have not yet become medullated, in the cerebrum the motor paths become ripe, apparently without exception, at a later period than the corresponding groups of sensory fibres, a fact which makes it seem probable that in the cerebral cortex the primary form of motor activity is the reflex.

One of the most pleasing anatomical results to which Flechsig has attained has been the accurate tracing out of the different sensory paths to their cortical end-stations, and of the motor paths passing corticofugally from these to motor neurones of the next order situated in the lower centres. For the first time he has succeeded in placing before us what appears to be almost a complete picture of

the various projection fibres of the cerebrum, both corticopetal and corticofugal.

Flechsig differentiates more or less sharply the sensory impulses which afford to the cerebral cortex information concerning changes going on in the body itself, and those which bring to it impressions from the external world. The former group of sensations includes the muscle sense, the various visceral sensations connected with many of the instincts, such as hunger, thirst, sexual desire, as well as many of the cutaneous sensations, while the other group comprises those received through the organs of special sense, the eye, the ear, the nose, the tongue and, in part, the skin. The first group, those which tell us about the condition of our bodies, are only in small part objective, on the other hand, those which tell us about the external world are always, or nearly always, associated in consciousness with the ideas of objects or processes outside ourselves. By this I mean that we project the latter sensations, but not the former, into the external world. The paths carrying these various sensory impressions to the cortex become medullated at very different periods. It is of no ordinary interest and importance to find that the fibres carrying impressions concerning what is going on inside the body are the first to become medullated; that is to say, it is the (indirect) continuations of the paths represented by the dorsal roots of the nerves of the spinal cord and medulla which first gain their myelin sheaths.

Beginning at the end of the eighth month of foetal life and progressing rapidly during several weeks following, medullated bands appear in what is known as the sensory region of the internal capsule, the posterior part of the posterior limb. At a time when all the rest of the cerebrum is entirely devoid of myelin, these medullated fibres, present in sections cut in suitable directions and stained by Weigert's method, appear as sharply circumscribed black bands running out from the internal capsule toward the central gyri. The distribution of the fibres isolated in this way affords a picture which is very striking when seen for the first time, and even to the naked eye is as clear and clean-cut as the lines of a diagram.

It has long been known from clinical and pathological experience that in the hemianæsthesia of Türck in which there is a disappearance of the cutaneous sensations and

muscular sense as well as of pain sensations from the oral cavity, the sexual organs, etc., the disease is localized in this region of the internal capsule. In some instances along with these symptoms the higher senses may be little or not at all affected; in others, as Türck himself observed, and as the studies of Charcot showed still more clearly, there may be associated anæsthesias of the higher senses of hearing, sight, etc. The simple form of Türck's hemianæsthesia, in which the impulses which are prevented from reaching the cerebral cortex are those which come into the nerve centres through the dorsal roots of the nerves of the spinal cord and medulla, can always be traced to lesions of the posterior part of the internal capsule and of the adjoining projection fibres.

Flechsig has made a careful analysis of the development of these tracts, and finds that the (indirect) continuations of the dorsal roots through the internal capsule can be divided into three definite systems, which become medullated at different periods and apparently subserve, in part at least, different functions. He has designated these three according to the order of their medullation as systems Nos. 1, 2 and 3. For system No. 1 the myelin appears at about the beginning of the 9th foetal month. It occupies in the internal capsule and in its upper half, the area immediately behind the fibres of the pyramidal tract. The fibres of this system in the main pass up from the basal portions of the lateral nucleus of the thalamus, the cup-shaped body (*schalenförmiger Körper* of Flechsig und von Tschisch) and in part directly out of the medial lemniscus. They are distributed exclusively to the cortex of the two central gyri, which are thus the first of all the regions of the cortex to become connected by means of medullated fibres with the sensory apparatus of the body. The fibres of this system are shown in the diagrams marked 1', 1''.

A few of the fibres of this system corresponding to the posterior angle of the lenticular nucleus run in the external capsule and in the most posterior part of the lamina medullaris externa of the lenticular nucleus. A small bundle appears to go into the lower part of the optic radiation (1+), the exact distribution of which is not yet certain.

The sensory system No. 2 begins to receive its myelin about a month later than does No. 1. The fibres of this system also go out of the lateral nucleus of the thalamus,

but more dorsally. A few of them issue from the centre médian of Luys. Passing upwards they are distributed in part to the central gyri, the lobulus paracentralis and to the foot of the superior frontal gyrus. Another part of them, after bending around at an acute angle and passing inwards, become distributed to the gyrus fornicatus along its whole length. The most posterior bundles (2', Fig. 1) enter into the cingulum and run toward the Ammon's horn. Still later another bundle belonging to this system runs from the lateral nucleus of the thalamus basalwards and enters into the uncus and arrives from in front and below at the subiculum cornu Ammonis. The whole of the limbic lobe thus comes to be connected with the lateral nucleus of the thalamus.

The third system, the last to become medullated, is also connected with the lateral nucleus of the thalamus, emerging from the anterior portion of it. It enters the internal capsule in about its middle portion and runs in part directly to the foot of the third frontal convolution, another part curving markedly as shown in the diagram (Fig. 1, 3, 3'') before reaching the cortex. Bundles of the latter run from the region of the pyramidal tract forward into the fasciculus subcallosus, and descend at the anterior margin of the corpus striatum to the third frontal convolution (3'). The fibres of a second group pass through the anterior limb of the internal capsule into the frontal lobe almost as far as the pole and then bend round at an acute angle, part of the fibres reaching the middle portion of the gyrus fornicatus (3), another part, the anterior half of the superior frontal gyrus, while single fibres go to the foot of the middle frontal gyrus.

The connections of the lateral nucleus of the thalamus to the parts of the nervous system below are, according to Flechsig, well understood. It is in this nucleus that one has to seek for the connections of the continuations of the dorsal roots, that is to say, of the medial lemniscus, the superior cerebellar peduncles and certain bundles of the formatio reticularis. This aggregate of sensory fibres under consideration corresponds to Flechsig's *Haubenstrahlung*. The cup-shaped body (*schalenförmiger Körper*) and the centre médian of Luys receive a portion of the sensory conductions of the dorsal roots; while the rest of the thalamus apparently has but little or nothing to do

with the conduction of these impressions. Flechsig, therefore, groups the lateral nucleus of the thalamus, the cup-shaped body (*schalenförmiger Körper*) and the centre médian together, designating them as the "ventro-lateral group of nuclei." In the developing baby the findings concerning the connections of these nuclei with the cortex agree with the results of secondary degenerations found at autopsy in the well-known case reported by Flechsig and Hösel, where both central gyri had been diseased for about fifty years in wide extent. In this case it may be remembered there had resulted secondary degenerations of the superior cerebellar peduncle, of the medial lemniscus and of a portion of the *formatio reticularis*, while at the same time the cup-shaped body and the lateral nucleus of the thalamus had undergone marked alterations with degeneration and destruction of their ganglion cells.

It is evident from what has been said that the cortical domains connected with the three systems of conductions under consideration are very extensive. The cortex involved takes in all that area extending from the posterior border of the posterior central gyrus forwards including on the external surface the two central gyri and the feet of the three frontal gyri; on the medial surface of the hemisphere it includes the lobulus paracentralis and the gyrus *fornicatus*. To the whole area Flechsig, adopting a term previously used by Munk, gives the name *Körperfühlsphäre*, which I shall, at the suggestion of Professor Gildersleeve, translate into English as the *somæsthetic area*.

The first of the paths connected with the organs of special sense to become medullated is that concerned in carrying olfactory impulses; the fibres of the olfactory tract obtain their myelin sheaths toward the end of the 9th month of foetal life and in the neighborhood of the path which they take, other systems of fibres are as yet, non-medullated, so that the destination of the former in the cortex can be made out with the greatest clearness and accuracy. The constituent fibres enter into relations with both the frontal and the temporal lobe. The fibres going to the frontal lobe are distributed to the whole posterior margin of its base as well as to the basal part of the gyrus *fornicatus*, while the majority of those distributed to the temporal lobe pass to the uncus and to part of the "inner pole" of the temporal lobe. By means of association paths

both the frontal and temporal portions of the olfactory cortex become connected with the Ammon's horn, and later other bands of fibres connect both olfactory areas of the cortex with the globus pallidus, the lenticular nucleus and the thalamus.

After the olfactory tract has become medullated the optic path gains its myelin. The fibres of the optic tract can be followed directly into the lateral geniculate body and to the superior colliculus of the corpora quadrigemina. According to Flechsig, the lateral geniculate body represents a place of relay in the optic path. The first portion of the optic radiation of Gratiolet to become medullated consists in reality of a bundle of fibres proceeding directly from the lateral geniculate body to the part of the cortex of the occipital lobe immediately adjacent to the calcarine fissure, and especially to that portion of the cortex which is characterized by the presence of the macroscopic stripe of Vicq d'Azyr.

In the new-born infant this "optic radiation in the narrower sense" is completely isolated, running past the thalamus, though not at all connected with it, to the occipital lobe. There is no proof, Flechsig holds, that in man the thalamus forms an internode in the path of impulses on their way to the visual area of the cortex. It is his idea that the "optic radiation in the wider sense," that is, in the sense of Gratiolet, represents much more than a simple visual conduction path. It is likely, he believes, that a large portion of this path is not corticopetal in conduction, but corticofugal. For the present, however, he names the whole cortical area, with which the "optic radiation in the wider sense" is connected, the visual sense area. This includes the whole of the inner surface of the occipital lobe, but on the convexity only a narrow zone in the region of the first occipital convolution and of the occipital pole, and not the external occipital convolutions or the angular gyrus. It is certain that within this domain the visual sense area in the stricter sense must be contained, but just how much of this region actually receives fibres which represent continuations (direct or indirect) of the optic tract is as yet, not certain. The facts which have been ascertained by those who have studied secondary degenerations agrees in the main with these developmental findings. In nodular disease in which only the region of

the calcarine fissure has been affected the portions of the white matter of the cerebrum which degenerate are precisely those which in the developing baby correspond to the "optic radiation in the narrower sense" which Flechsig describes, and, with the degeneration of this radiation, the lateral geniculate body is destroyed also. This makes it extremely probable that the regions distant from the calcarine fissure can have only a very limited, if any, part in the *direct* reception of visual impulses.

Further evidence in favor of this view of the distribution of the conducting paths to such a circumscribed area is to be found in the results of clinical observation. Hemianopsia due to cortical disease occurs only when the inner surface or the pole of the occipital lobe is affected. Nothnagel pointed this out as early as 1887 and subsequent writers agree with him. Lesions of the angular gyrus so long as they do not go deep enough to affect the optic radiation lying in the white matter beneath lead to no defects in the visual field, a fact which Flechsig believes proves the incorrectness of the views held by Ferrier regarding the position of the visual sense area in man.

The fibres conducting impulses concerned in sensations of taste and their cortical connections have not yet been localized. It is probable that the area concerned does not lie far from the somæsthetic area or from the olfactory sense area.

The fibres conducting auditory impulses, that is to say, the indirect continuation upwards of the cochlear nerve are the last of all the sensory fibres in the brain to become medullated. As has been known for some time the impulses arriving along the cochlear nerve pass onwards mainly by means of the lateral lemniscus which is intimately connected with the inferior colliculus of the corpora quadrigemina and with the medial geniculate body. Von Monakow has proven that the latter is connected by means of projection fibres with the cortex of the temporal lobe and exclusively with this lobe. Flechsig has shown that in the medullation of the white matter of the temporal lobe it is the auditory path connecting the medial geniculate body with the cortex, which first obtains its myelin. This band of fibres becomes medullated at a period when all the rest of the white matter in this region of the temporal lobe is still unripe. These fibres terminate in the two transverse

gyri of the temporal lobe, preferably in the anterior, two gyri to which hitherto little attention has been paid since they lie buried in the depth of the fossa Sylvii. They are connected with a portion of the superior temporal gyrus,

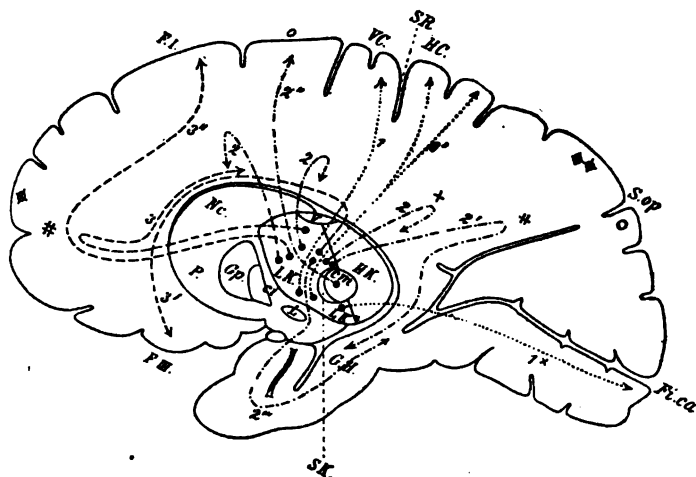


Figure 1. Sagittal section through the human brain. (After Flechsig) Gp globus pallidus of the lenticular nucleus; P putamen; Nc nucleus caudatus; LK lateral nucleus of the thalamus; SK cup-shaped body of thalamus (schalenförmiger Körper); cm centre median of thalamus; HK medial nucleus and pulvinar; v anterior nucleus of thalamus; LK, SK, cm, together represent Flechsig's ventro-lateral group of nuclei of the thalamus; HK, v represent his dorso-medial group of nuclei; ci internal capsule; L Luys' body; Fi superior frontal gyrus; Fiii inferior frontal gyrus; GH gyrus hippocampi; VC anterior central gyrus; HC posterior central gyrus; SR sulcus centralis Rolandi; S. opersculus occipito-perpendicularis; Fi. ca fissura calcarina; 1, 1', 1, 1''' sensory system No. 1; 2, 2', 2'' 2''' sensory system No. 2; 3, 3', 3'' sensory system No. 3; different kind of dotted lines are used to represent these three systems in all the figures. The corticopetal paths of the optic thalamus are represented in the figure; the corticofugal conductions of the dorso-medial group of nuclei of the thalamus, the motor paths of the cerebral cortex, etc., are not shown. The arrangements of the points in the ventro-lateral domain of the thalamus is schematic.

and this is of very great interest since it is exactly this portion of the superior temporal convolution (namely its third and four-fifths reckoned from its anterior extremity) which Naunyn has shown to be most often involved in the perceptive form of word-deafness, a variety of sensory aphasia

which Wernicke had earlier successfully localized in the superior temporal convolution of the left side.

There can be no doubt, therefore, that the cortical region concerned represents the auditory sense area, especially since v. Monakow (Arch. f. Psych. Bd. XXVII.) has shown that with the destruction of this region the medial geniculate body eventually degenerates over its whole cross section. The clinical observation that with one-sided cerebral disease there is scarcely ever complete deafness of the opposite ear, is explained by the well-known partial decussation of the cochlear nerve in the pons and in the inferior colliculi of the corpora quadrigemina. Whether or not the vestibular nerve is connected in any way with the auditory region of the cortex has not yet been made out.

Flechsig does not deny that the centres below the cortex may not entirely be excluded from mediating sensation, that is to say, of consciously perceiving to a limited extent. But if there be such a subcortical consciousness it must, in his opinion, be concerned with extremely vague and unlocalizable sensations.

But even when all the sense areas of the cortex, extensive as they are, have become medullated, only about one-third of the surface of the cerebrum has been involved. The individual sense-areas are isolated, not touching one another directly. They are separated by regions which have no direct connection at any rate at this stage of development with the centres below or with the periphery of the body. It will have been noticed that the somæsthetic area, that portion of the cortex at which bodily sensory impulses arrive, includes within it what has usually been described as the motor zone of the cortex. In all probability also from the visual, from the auditory and from the olfactory sense areas, corticofugal, probably motor, conductions also go out. Indeed, Flechsig has already been able to throw much light upon the extent and distribution of such conducting paths. The nomenclature adopted for the sense-areas is to be regarded, therefore, as Flechsig states, only as holding good *a potiori*. In reality each area is not simply sensory, but sensory-motor, but nevertheless the most convenient way to designate them, is after the qualities of sensations of which they represent the internal sense-organs.

Passing out from the somæsthetic area, after the sensory fibres of system No. 1 have been medullated, on each side is found that large mass of fibres which taken together constitute the pyramidal tract. This bundle of fibres appears to be destined to innervate all the muscles of the body concerned in the finer tactile movements and would

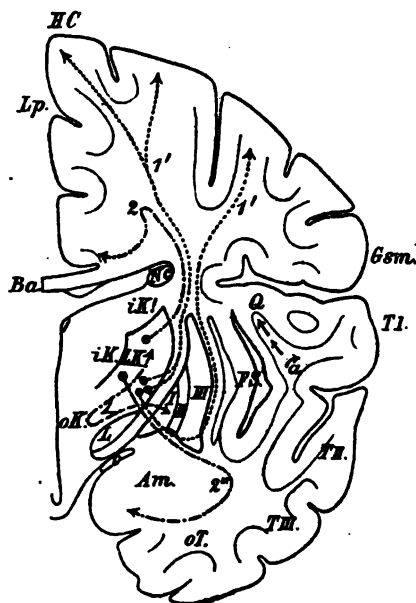


Figure 2. Frontal section through the human brain. (After Flechsig) I, II, III First, second and third portion of the lenticular nucleus; LK lateral nucleus of thalamus; iK, iK' medial nucleus of thalamus; Nc nucleus caudatus; L Luys' body; oK brachium conjunctivum; o tractus opticus; Am nucleus amygdalæ; FS Fossa Sylvii; HC posterior central gyrus; Gsm gyrus supra marginalis; Ti, Tii, Tiii superior, middle and inferior temporal gyri; Q anterior transverse temporal gyrus; oT gyrus occipito-temporalis; Lp lobulus paracentralis; Ba corpus callosum; a auditory conduction path.

seem to be the only path running directly from the cerebral cortex to the cells of origin of the motor nerves of the medulla oblongata and spinal cord. It is this path which in one sense may, therefore, be regarded as the one concerned in placing the organs of the sense of touch under the domain of the will.

Corresponding to the system of sensory fibres designated as No. 3, and taking their origin in the corresponding portion of the somæsthetic area, is the frontal cerebro-corticopontal path which runs corticofugally. It passes through the base of the cerebral peduncle forming the medial fibres there, and terminates among the ganglion cells of the pons. This frontal cerebro corticopontal path is concerned, Flechsig believes, with the movements of bilaterally innervated muscles, such as those of the eyes, neck and trunk muscles which are not under the control of the neurones whose processes correspond to the axones of the fibres of the pyramidal tract. It is considered probable that the motor impulses concerned in the speech-movements are also carried down by the frontal cerebro corticopontal path. Whether in the foot of the peduncle there is a motor correlate for the sensory fibres of the somæsthetic area belonging to system No. 2, Flechsig has not been able to determine. Certain it is that from corresponding portions of the cortex of the somæsthetic area to the thalamus there run an enormous number of corticofugal fibres, but if these can influence parts below, they probably do so by means of secondary centrifugal paths which start out from the thalamus and pass downwards through the tegementum. The descending corticothalamic paths generally are connected in the thalamus not with the ventro-lateral group of nuclei mentioned before, but rather with the portions of the thalamus so far not shown to be connected with the indirect continuations upwards of the sensory fibres of the posterior roots (*Haubenstrahlung*); that is to say they are connected with the anterior nucleus, the internal or medial nucleus and the pulvinar, a group of nuclei which Flechsig designates in contrast to the other as the "dorso-medial group."

Not all parts of the "dorso-medial group" appear, however, to be connected with the somæsthetic area. Each portion of the former apparently stands in connection with a special cortical domain. Thus the anterior nucleus of the thalamus has, as its cortical representative, the limbic lobe, the medial nucleus in its lateral and dorsal part is connected with the central gyri and in its medial part with the feet of all the frontal convolutions and the corpus striatum. The pulvinar apparently has no connection with the somæsthetic area, but receives corticofugal paths from

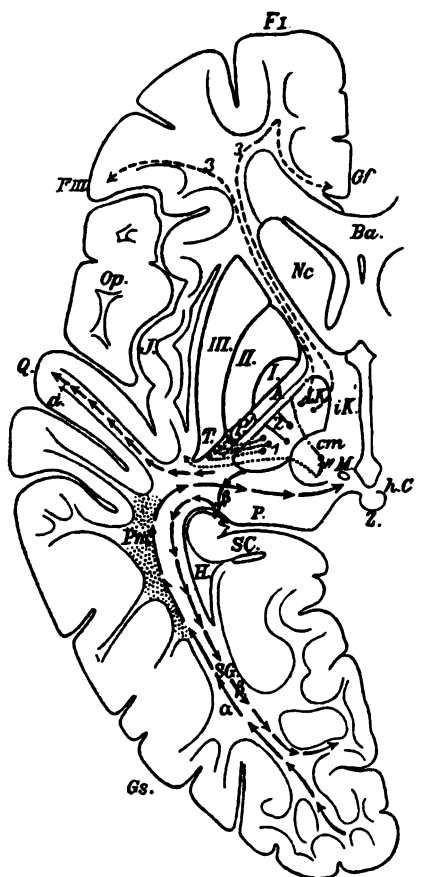


Figure 3. Horizontal section through the human brain (After Flechsig.) I, II, III, first, second and third portion of the lenticular nucleus; Nc nucleus caudatus; LK lateral nucleus of thalamus; iK medial nucleus of thalamus; cm centre median; P pulvinar; M Meynert's bundle in cross section; hC posterior commissure; Z pineal body; P' pyramidal tract; A Arnold's bundle of internal capsule; T sensory region of internal capsule; a auditory conduction path; SG Gratiolet's radiation ("Optic radiation in the wider sense"); a cortico-fugal paths of Gratiolet's radiation; B corticopetal paths of Gratiolet's radiation projection fibres of the lateral geniculate body; Q anterior transverse temporal gyrus going over into the superior temporal gyrus; Gs gyrus subangularis; Fi superior frontal gyrus; Fiii inferior frontal gyrus; Gf gyrus fornificatus; SC subiculum cornu Ammonis; H posterior horn of lateral ventricle; Op operculum; Pm (dotted) cross section of large association system between somæsthetic area (central gyri) and posterior large association centre; J cortex of island of Reil.

the visual sense area, and perhaps in its anterior portion from the auditory sense area.

The most of the fibres of a special motor system pass out from the auditory sense area and its immediate neighborhood to become connected lower down to the mass of ganglion cells in the pons, especially to its distal portions. This is Flechsig's temporal cerebrocorticopontal path and makes up in the main, he thinks, that portion of the base of the peduncle lateral from the fibres of the pyramidal tract. (Türck's bundle, Meynert). With total destruction of the auditory sense area on one side, however, this bundle does not degenerate entirely; a small number of the fibres, the origin of which is not yet clear, remain unaffected. So that leaving out of consideration these few and the bundle of sensory fibres of the base (Fussschleife), the whole of the fibres constituting the base of the peduncle can be accounted for by (1) the pyramidal tract; (2) the frontal cerebrocorticopontal path, and (3) the temporal cerebrocorticopontal path, that is to say, by motor paths throwing motor neurones of the pons, medulla and spinal cord under the governing influence of certain of the sensory-motor areas in the cerebral cortex. The corticofugal paths from the visual sense area probably run in the "optic radiation in the wider sense," that is, in Gratiolet's radiation.

As far as Flechsig has been able to make out all or very nearly all of the projection fibres of the cerebrum are accounted for when the corticofugal and corticopetal paths of the different sensory-motor areas, as outlined by him, are summed up. But, as I have said, nearly two-thirds of the whole of the cortex yet remains to be accounted for. What is the significance then of the regions which are not provided with projection fibres? The studies of Flechsig have thrown these portions, which in large part correspond to what we have been accustomed to call the "silent areas" of the cerebral cortex, into bold relief. His anatomical investigations, especially when their results are compared with the findings of pathological anatomy in cases which have been carefully studied clinically, indicate that these hitherto insufficient explored regions possess functions of the greatest importance and interest.*

*Thomas, speaking in this connection, says, "Flechsig's association-centres are what have been called the silent areas of the brain:

If you will recall the limits described for the different sense areas it will be obvious that the regions left over include in the frontal lobe the anterior portions of the superior and middle frontal convolution portions of the inferior frontal convolution and the gyrus rectus; further, the greater part of the island of Reil, the first and second parietal convolutions, the middle and inferior temporal convolutions exclusive of the internal temporal pole, the occipito-temporal gyrus and the whole of the occipital lobe not included in the visual sense area. In the diagram before you these relations are shown very clearly. (Figs. 4 and 5.) The sense areas are dotted, the regions in which the majority of the sensory fibres terminate being closely studded with dots, while in between the different sense areas the large undotted regions correspond to the portions of the cortex entirely devoid of projection fibres, or at most provided with extremely few of such fibres. At the periphery of each sense-area is a marginal zone in which projection fibres are less thickly distributed.

The white matter corresponding to all the cortical regions between the sense areas with the exception, perhaps, of the subangular gyrus becomes medullated considerably later than that of the sense centres; so that even in children, three months old, the former are sharply distinguishable from the latter by their poverty in myelin. Flechsig finds, however, that medullated paths gradually grow out from the sense centres into these non-medullated regions. Further, between the individual gyri of the non-medullated regions, bands of association fibres gradually ripen, connecting the single gyri with others near them and also with gyri at a distance. By means of the corpus callosum the gyri in one hemisphere are connected with those of the opposite hemisphere. Flechsig, on account of the marked predominance of association systems in these areas has designated them "association centres of the cerebral cortex." He does not as did Meynert, believe that the individual sense centres are connected directly with one another, but thinks that on the contrary, they are connected

but we are forced to believe that they are silent, not because they do not speak, but because we are too dull of understanding to hear what they say." Cf. Thomas, H.M., and Keen, W.W.—A Successful Case of Removal of a Large Brain-Tumor. *Am. Jour. of Med. Sciences*, Nov., 1896.

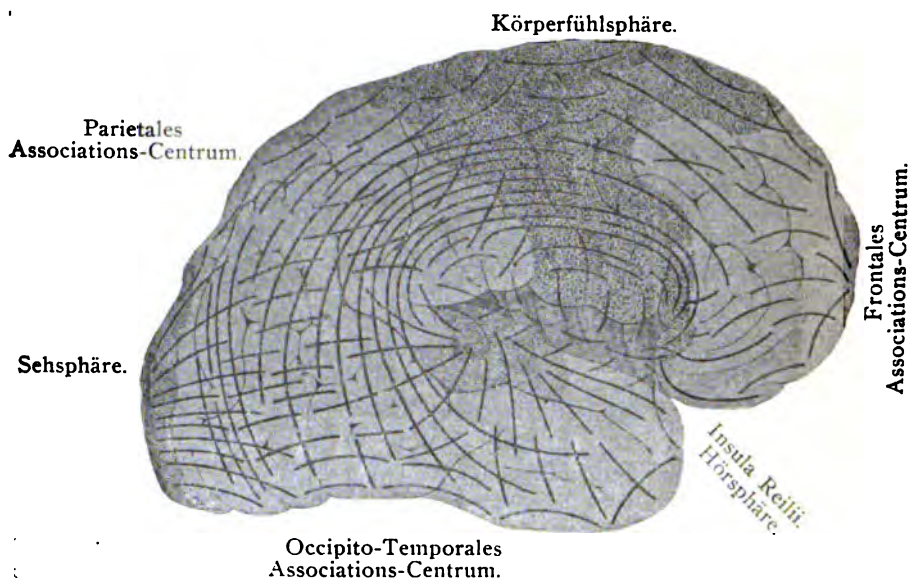


FIGURE 4.

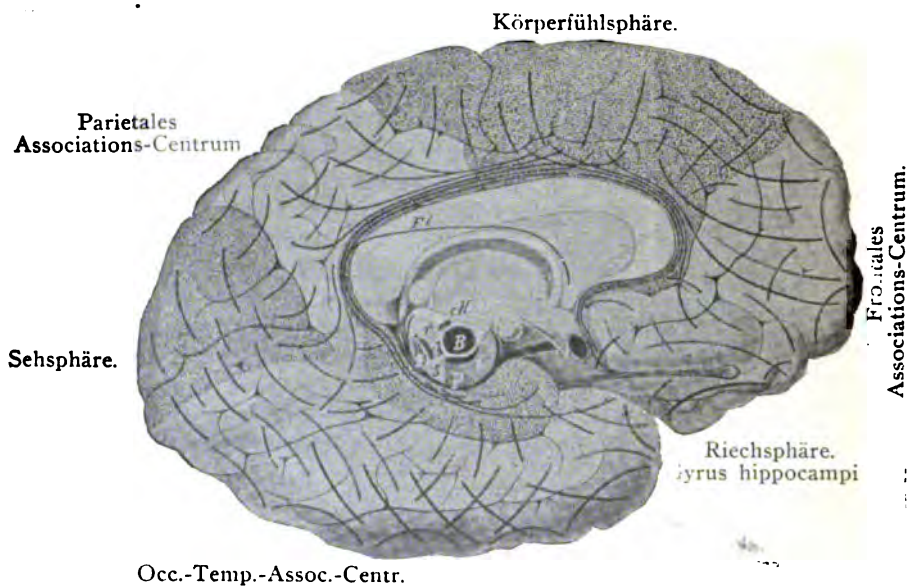


FIGURE 5.

Figs. 4 and 5. External and internal surface of cerebral hemisphere showing sense centres and association centres (after Flechsig).

B brachium conjunctivum (red nucleus); c medical lemniscus; r formatio reticularis; cH central tegmental path; P pyramidal tract; 5 temporal cerebrocorticopontal path; 6 frontal cerebrocorticopontal path; g medial geniculate body.

Instead of the German terms in large letters on the plate the following English equivalents (in brackets) are to be used:—Körper-fühlspähre (Somæsthetic area); Parietales Association-Centrum (Parietal association centre); Sehsphäre (Visual sense-area); Occipito Temporales Association-Centrum (Occipito-temporal association centre); Hörsphäre (Auditory sense-area); Insula Reilii (Island of Reil); Frontales Association-Centrum (Frontal association centre); Gyrus Hippocampi (Gyrus Hippocampi); Riechspähre (Olfactory sense-area).

The lines drawn upon the cerebral convolutions indicate in all instances association systems.

only indirectly, by means of the association centres. The latter receiving conduction fibres from adjacent sense centres and from adjacent as well as distant association centres, furnish an anatomical mechanism which makes possible the working up into higher units of simple sense impressions and of combinations of simple sense impressions of the same quality and of different qualities. Thus, Flechsig denies the function ascribed by many to the so-called fasciculus longitudinalis inferior which would make it a system associating directly two sense centres with one another. He thinks that on the contrary it has an entirely different significance which, however, I shall not discuss at this time.

The position of the individual areas of association properly throws some light upon the functions which they subserve. Thus, the large region which Flechsig designates as the posterior large association centre and which includes the præcuneus, all the parietal convolutions except the posterior central gyrus, part of the gyrus lingualis, the spindle-shaped gyrus and the middle and inferior temporal gyri as well as all portions of the three occipital gyri not concerned in the visual sense area, is situated between the visual, the somæsthetic and the auditory sense areas. The island of Reil is surrounded by the somæsthetic area, the auditory area and the olfactory area, and into it run bands of fibres from these sense areas, so that it, Flechsig thinks, is properly designated as the middle association centre. The main portion of the frontal lobe,

Flechsigs's anterior association centre, is very intimately connected with the somæsthetic area, and with the olfactory sense area. As Flechsigs remarks, however, it is not probable that its function is confined to the association of olfactory impressions, with sensations which tell us about the condition of our bodies, for the olfactory sense in man is relatively little developed, while the frontal lobe is developed *ad maximum*.

While the anatomical evidence, which Flechsigs has brought forward, would seem to be sufficient to indicate in general the essential nature of the functions of the different regions of the cortex described, there has been already collected a great mass of clinical and pathological data which when reviewed in the light of the newer anatomical knowledge affords confirmatory proof of his views in a very surprising manner. Lesions involving the sense centres are followed by a train of symptoms of an entirely different character from those which accompany lesions of the association centres. This will be immediately clear when I recall certain examples which are doubtless already familiar to you. All the evidence goes to show that the phenomena of sharp, clean-cut sensations as they appear in consciousness have their origin in the sense centres. As a result of a lesion of a given sense centre this sharpness of sensation for the particular sense quality concerned disappears. If the visual area on both sides of the brain be destroyed, the patient no longer sees. He may occasionally believe that he still possesses visual sensation, but on closer examination it will be found that the picture in his consciousness is a memory-picture, not a perception. Patients suffering from double-sided destruction of the auditory sense area are absolutely deaf, and it is to be imagined that if both somæsthetic areas were entirely destroyed the individual would be deprived of sensations informing him of the condition of his body, of the skin and of the viscera. Injury to the central gyri on the left side about their middle has been followed by loss of power to recognize correctly the form of a given object when the right hand alone has been used to feel it. Irritation of the posterior central gyrus may lead an individual to believe that he experiences movements of his thumb even when his eyes convince him that it has not moved. Tumors pressing upon the olfactory sense area have given

rise to subjective odors, while mental images of colored figures and the like have occurred in individuals in whom after death cysts involving the visual area of the occipital lobe have been found. Irritative lesions of the auditory sense area can give rise to noises and to other sound-images. Destruction of the sense centre concerned with the sensations of any given sense-quality is associated with the so-called perceptive sensory disturbances.

The sense centres are concerned not only in the bringing into consciousness of the individual elementary sense impressions; but the distinction of the spatial and temporal relations of these impressions, if Flechsig be right, is also to be attributed to the activities of the cortical sense spheres. Thus, "perceptive word-deafness" has been shown to depend in right-handed individuals upon disease of the left auditory sense area. When words are spoken the individual hears only confused noises and does not understand them. Wernicke believed that in such cases there is a permanent loss of the memory pictures of the sounds of words. This is denied by Flechsig, who holds that here we have to deal rather with an incapacity of separating the sounds which follow one another in the spoken words, of distinguishing tone intervals correctly between syllables and words. The patient does not perceive an orderly combination of sounds, but an undecipherable chaos of tones and noises. If in such cases we have to deal with pure lesions of the auditory sense area the patients can speak spontaneously a large number of words correctly, so that the ordinary observer might scarcely notice any disturbance in his speech, a fact which proves that the memory pictures of the sounds of words have been retained in spite of the destruction of the auditory sense area. On the other hand, if the periphery of the auditory sense area be destroyed, as in Heubner's well-known case, and the auditory sense area itself remain uninjured, the clinical picture is exactly reversed; the patient in this transcortical sensory aphasia (in the sense of Lichtheim and Wernicke) can speak spontaneously only very few words (amnesic aphasia) or there exists a high degree of paraphasia. The patient, however, can from the beginning repeat correctly words which are spoken before him, a proof that he has heard the words correctly and has retained the capacity for perceiving correctly the intervals between syllables

and words. If, in spite of this, word-deafness exists, the reason lies in the fact that the words correctly heard do not call forth by association into consciousness the memory pictures which belong to them and which help to make up the sense ("apperceptive word-deafness" of Herbart). Of course, these forms are seldom met with pure, for in the majority of cases the auditory sense area and its periphery are diseased more or less together and mixed forms are of no use for the decision of the question whether and in how far diseases of the auditory sense area alone have disturbances of memory as a result.

If then the cortical form of "perceptive word-deafness" does not depend on the loss of memory pictures of the sounds of words it is highly probable that it is a "sensory atactic" disturbance—the temporal arrangement of the auditory sensations is lacking. If Flechsig be correct about this then the essential basis for spatial and temporal perceptions is to be sought in the sense spheres. This necessitates the supposition that the neurones of the sense centres possess a certain kind of memory, the capacity, for example, of holding a tone in the memory until the word or the sentence is at an end. The sense spheres in man appear, however, to be incapable of reproducing independently large numbers of memory pictures, and any extensive loss of this power is dependent upon diseases localized outside the sense spheres. The fact that cases have been observed in which the reproduction of visual impressions have suffered little, even when both visual sense areas have been destroyed, has led clinicians some time since to the assumption that optic memory pictures and visual sensations must be attached to separate cortical domains.

Flechsig thinks that the tactile disturbance, described by Wernicke, associated with disease of the somæsthetic area is due to loss of the capacity of uniting properly tactile stimuli to a mental image (that is to say, the spatial arrangement of the single impressions is no longer possible). It is rather an atactic disturbance of perception than, as Wernicke thinks, a defect of memory-capacity. It is quite analogous to the perceptive word-deafness dependent upon lesion of the auditory sense area. It is probable that the organic traces of the more extensive memory-pictures built up of no matter what sense qualities are as-

sociated with the cells of the association centres which lie between the sense centres.

Before passing to the description of the functions of the association centres, however, a few special points in connection with the sense centres must be considered. With regard to the somæsthetic area it has long been known that disease of the central convolutions is accompanied frequently with loss of the kinæsthetic organ-sensations, so that the mental images of position and movement for the extremities and the region of the mouth may be absent or defective. Along with these symptoms, especially where the foci of disease are small the cutaneous sensations suffer only as regards the tactile sense and its exact localization. As a result of lesion of the arm region there is an incapacity to recognize external objects by feeling their form.

If the inferior frontal convolution be diseased, the capacity for calling up images of movements or rather the capacity to feel the position of the organs which participate in speech suffers so that system No. 3, connected with the inferior frontal gyrus, is accordingly not different in the sensation-quality mediated from the sensory paths of the central gyri, but simply in regard to the region of the body whence the sensations come; the new-born infant for purposes of self-preservation makes use far earlier of his extremities, his lips and his tongue than of his trunk and speech muscles, and this fact of experience agrees extremely well with the developmental finding that the sensory and motor paths of the extremities develop earlier than those for the trunk and the special organs of speech. Certain of the fibres of system No. 2 have apparently to do with muscle sense, but our knowledge is as yet insufficient concerning this group. There is a good deal of evidence, too, that the somæsthetic area plays an important part in the coming into consciousness of many of the bodily processes accompanying or constituting the emotions and that thence start out many of the movements which serve as the expression of the emotions, a point of view which if confirmed is of infinite importance for psychiatry.

Let us now turn to the subject of the functional activities of the association centres. We have seen before that from the anatomical arrangement, these areas appear to exist for the purpose of uniting the activities of the var-

ious sense centres. Clinical and pathological evidence, too, is in favor of Flechsig's view that they are concerned in the higher manifestations of the intellect, in the processes of memory, recognition, judgment and reflection. It is in disease of these areas that we see, above all, disturbances of memory and of the associative processes. When the posterior large association centre, for example, is diseased, the lesions are not accompanied with phenomena of perceptive deafness, of perceptive blindness, or of perceptive tactile anæsthesia, provided the adjoining sense centres remain unaffected. But instead of these an entirely different group of clinical phenomena becomes manifest. Here we meet sometimes with the conditions known as mind-blindness, mind-deafness and the like; with apraxia or agnosia; sometimes there is weakening of the power of visual imagination. There may be an incapacity to call into consciousness melodies which the individual formerly knew well, and in lesions of this area on the left side in certain portions sensory (optic) alexia, optic aphasia (amnesic color blindness), apperceptive (transcortical) word-deafness, verbal paraphasia, and sensory amnesic aphasia (incapacity to call up the memories of the sounds of words corresponding to the mental images in consciousness). The memory-capacity may in such instances be affected apparently in either or both of two ways: (1) by destruction of the association paths concerned in setting free given mental images, and (2) by actual, permanent destruction of the organic memory traces in the nerve-cells. It would appear, therefore, that the posterior large association centre is concerned in the formation and collection of ideas concerning the external world; that is of actual knowledge concerning external objects, of combinations of sounds, and the union of all these with one another. In these regions are stored up the elements of our positive knowledge as well as the factors which come into play in the exercise of the phantasy. It is here that preparation is made for speech which shall accord with the thoughts; in short, this region more than any other in the cerebral cortex appears to be the site of the processes concerned in what we ordinarily mean when we speak of the "intellect."

The anterior association centre, that is the association centre of the frontal lobes, has manifold connections with

the somæsthetic area and hence also the motor regions concerned in conduct. So that here in all probability, Flechsig states, is to be sought the anatomical mechanism by means of which memory-traces of all conscious bodily experiences, especially of acts of the will, are stored up. The study of the functions of this region of the brain is extremely difficult, and as yet only general statements can be made regarding them. It would appear that the positive knowledge of the individual concerning external objects does not necessarily suffer in diseases of this portion, at least at first, although the appreciation of the value of this knowledge and its relations to the individual himself may not be recognized. The man may lose interest in the external world as well as in himself and cease to participate personally in what is going on about him. Indeed it is in the diseases affecting this area and the neighboring somæsthetic area that most marked alterations in the character of the individual are met with. The phenomena of attention, of reflection and of inhibition are possibly especially connected with this frontal association centre. Wundt has for some time believed that the "active apperception" is to be localized in this region.

If Flechsig be correct in his views it is evident that the study of the normal functions of the association centres is of the highest importance and will in the future represent preeminently the task of psychology; while the phenomena which result when the association centres are diseased will afford the especial topic of investigation for psychiatry. The study of cases in the literature, more especially of general paresis, where careful pathological examinations have been made after death has already thrown considerable light upon the function of these areas. Of course, in the majority of cases of this disease the lesions involve very different regions of the cortex at the same time. While in some instances, however, the disease has affected preferably the association centres alone without involving the sense centres, in a few cases the frontal association centres alone have been the ones chiefly involved, and in others the large posterior association centres have been the ones mainly affected.

Such material permits of an analysis of the functions of the individual areas. Thus, where the frontal lobes on

both sides of the brain have been diseased, the main symptoms recognizable during life have been those referable to an alteration or loss of ideas regarding the individual's personality, and his relations to what is taking place inside and outside his body, symptoms which are highly suggestive when compared with the results of extirpation of the frontal lobes in higher apes as carried out by the Italian investigator, Bianchi. The symptoms may vary much, probably according as the lesion is irritative or destructive in its nature. Thus, in some instances, there is an over-appreciation of self. The patient's egotism is unbounded. All things are possible to him. He is a multi-millionaire, a genius, or a high dignitary. In other cases he shows remarkable self-depreciation, and lack of confidence in personal capacity. The speech may for a long time remain unaffected, but the capacity for judgment, as to what is right and what is wrong and what beautiful and what hateful, is often involved so that the individual will exhibit in his conduct characteristics entirely incompatible with what his friends knew of him earlier in his life. Such persons lack self-command, even when uninfluenced by violent emotions, and when they are exposed to unusual stimulation, to anger or to sexual excitement, they lose all control of their conduct and are guilty of outrageous acts. Finally, if the disease progress far enough, imbecility appears and the individual may lose completely his ideas concerning his personality.

When the posterior large association centres have been mainly affected the clinical picture is very different; in these cases it is the knowledge of the external world rather than that of his body and of his personality which is defective, just as one would expect from what has been said above concerning the phenomena of sensory aphasia met with in focal softening of the cortex due to vascular disease. In these individuals the ideas regarding the personality may be tolerably clear; they may have almost perfect self-possession, but their friends notice and they may themselves be aware that they are unable to recognize objects seen and felt or to associate the elementary sensory impressions with the memories of experiences in their past lives. Such a patient will name external objects wrongly, misunderstand their use, confuse persons, and be mixed up in his ideas of time and space. He is unable to put into

words the images which float in his consciousness and suffers on the whole from a poverty of ideas. Yet with all this he may perhaps have a normal regard for himself and for his friends.

With combined diseases of the different association centres and especially with combinations of disease of the sense centres with disease of the association centres, the possible variations in the clinical picture become enormous. For the analysis of these symptoms and their anatomical localization psychiatry has been provided in these researches of Flechsig, should they be confirmed, with a most important aid.

It will be of especial interest to study the functional diseases of these different areas, disturbances of a temporary nature which can be ascribed to faulty metabolism, in the different areas dependent upon various factors such as imperfect nutrition, certain intoxications, prolonged emotion, excessive mental and physical activity and the like. The protean symptoms of neurasthenia and hysteria often in individual cases bear a special stamp which may enable us in the future to suggest with some probability the portion of the brain mainly responsible for their appearance.

Representing as they do ideas which fundamentally affect our general concept of the structure and function of the brain, these researches of Flechsig have, as might have been expected, not passed unchallenged. After his address at Frankfurt last fall a number of leading neurologists and psychiatrists discussed his findings and his views. It may be interesting to consider briefly some of the objections which have been offered to them.

A number of investigators are unwilling to grant that the areas of the cortex to which projection fibres are distributed are as limited as Flechsig would have us believe. Thus, von Monakow asserts that projection fibres go to nearly all parts of the cortex, though certainly some parts of it receive fewer by far than others. Hitzig, too, grants that the number of projection fibres going to the frontal lobe is very small. Von Monakow bases his objection upon the results of his studies of secondary degenerations. He finds degenerations in the thalamus after lesions of certain of the regions falling within the domains which Flechsig calls association centres. He believes, too, that

motility and sensation are represented in the cortex fundamentally differently from one another. His studies have convinced him that the sense areas occupy much more extensive fields of the cerebral surface than those indicated by Flechsig in his diagrams. Thus, the area for cutaneous and muscular sensations von Monakow thinks extends far beyond the central gyri; since to produce atrophy of the lemniscus and of the nucleus funiculi gracilis and the nucleus funiculi cuneati of the opposite side, destruction of the cortex (in both animals and man) of a far greater extent than that which represents the "motor zone" must have preceded. In answer to this Flechsig suggests (1) that a totally insufficient amount of material has been studied by the secondary degeneration method to afford conclusive results; and (2) that in many instances not sufficient attention has been paid to the exact localization of the lesion; that is to say not enough care has been taken to determine whether it has been purely cortical or whether it has involved also the sub-cortical white matter. He points out, for example, that lesions of the parietal cortex have been followed in a number of instances by degenerations of projection fibres, but in all such instances he believes the cortical nodule has affected bundles of projection fibres belonging to other parts of the cortex, but situated beneath the area diseased. The results of experimental degenerations in animals following extirpation of cortical zones cannot properly be directly applied to human beings, for in man there is a development of the association centres not reached in the brain of any other animal.

Another objection which very properly has been offered by Sachs and others is this: That after a certain period of development the medullation has become so diffuse in the cerebrum that it would be impossible to deny that late projection fibres passing to the association centres may become medullated. It must be granted that Flechsig can claim the limitation of sense centres as he defines them only for a definite period of development. It is certain, however, that at this period the primary sense centres are sharply marked off from the rest of the cortex.

Von Kölliker's objection to designating the association centres as intellectual centres is based upon his view that there is no essential difference between the pyramidal cell

of the various regions of the cortex. In the first place, however, as Flechsig points out, the time is not yet ripe for the building up of a psychology based upon the histology of the cortical cells. One need only refer to the futile attempt which has recently been made by Ramón y Cajal. As a matter of fact, however, the sense centres do differ very essentially not only in the correlation of the elements present in them, but also in the actual shape and position of the individual nerve cells. A skilled histologist who has studied sections from these regions can easily distinguish a section from the middle part of the gyrus fornicatus from one taken from the neighborhood of the calcarine fissure, from the middle of one of the central gyri or from the angular gyrus.

The students of purely introspective psychology have naturally always looked askance at contributions made to the subject of the mind by anatomists and by medical men in general. When we consider, however, the relatively little advance which has been made by purely introspective methods in late years and of the enormous help which physiological experiment, anatomical study and pathological observation have afforded, medical men will not be disturbed by the opposition of the metaphysicians. Nor need we be alarmed that certain objectors see in these newer anatomical and physiological studies a tendency to a return to phrenological views. Should they bear any resemblance to such doctrines this would by no means expose them to the curse of ridicule.

These studies of Flechsig taken together with the researches of Edinger show that the anatomical mechanisms underlying the mental processes in human beings as well as in animals are organically membered, and are only secondarily fused together into an organic whole. From the study of the gradual development of the individual organs of the brain as shown by ontogenetic and phylogenetic investigations, we have the promise of a clear and sharply defined picture of the various anatomical substrata which in definite sequence are concerned in the gradually increasing complexity of the organizing intelligence. While it is probable that many of the theories which go far beyond actual findings which Flechsig has advanced regarding psychology will with further knowledge be entirely given up or much modified,

still every one who reads his papers carefully will be ready to grant that many of them are too well founded to be overthrown. At any rate, he has supplied us with a mass of material and data which must form the starting point of a whole series of subsequent investigations.

In all his communications Flechsig has laid stress upon the predominating importance of the somæsthetic area. The largest in extent of all the sense centres, representing probably not a single centre, but a large group of individual centres, this portion of the cerebral cortex stands in a peculiar relation not only to the organs of the body, but also to the organs of the mind. It is on the one hand, the medium by means of which all the bodily occurrences, the bodily emotions and the bodily needs are brought into consciousness, and the region of the brain whence start out nearly all the motor impulses concerned in the conduct of the individual. On the other hand, it is connected intimately by means of association paths with the large association centres of the cortex; in front with the frontal association centre; behind with the large posterior association centre. On considering these associative relations it may be looked upon as forming the actual middle point of the organ of the mind. It is infinitely richer in association systems than any of the other sense areas. It is thus conceivable that the somæsthetic area has by far the greatest significance for the "awake" condition. Placed as it were, midway between the organs of the body and the organs of the mind it represents, Flechsig thinks, a sort of wrestling ground where the lower bodily instincts struggle for the mastery with the higher feelings and ideas. It is the first of all the sense areas of the cortex to become functional, and throughout life it would appear to take the lead in the intellectual processes. Without it, one can scarcely think of the possibility of the development of an intellectual personality, while we know from Laura Bridgeman's case that the auditory area, the visual area and the olfactory area can be absent, not only singly, but all together without interfering with the existence of a relatively good intellectual functional capacity.

It is evident from these embryological studies that the developing individual obtains first impressions from its own body, and that only later are there added to these the impressions from the external sense organs; so that

instead of thinking of an equivalence among the sense areas we have to consider the existence of subordinate relations. The mental organ is to be looked upon rather as a monarchy than as a republic, since the somæsthetic area is the main bearer of the self-consciousness throughout life, and since out of it go the majority of the motor paths concerned in conduct. According to Flechsig, therefore, the structure and the integrity of this centre are of the greatest significance for the character of the individual.

The relation of these studies to ethical problems and to the ultimate problems of life and character need not here be dwelt upon. Since we know that the health of the neurones in the cerebral cortex is essential for the proper inhibition of the lower centres concerned with the more primeval instincts and emotions their ethical bearing is obvious. When the intellectual centres are paralyzed, and especially when there is wide-spread disease of the somæsthetic area on the two sides, there often results most marked disorganization of the mental processes and most serious alterations in the character of the individual. The struggle between the lower instincts and the ethical feelings may cease, and instead of a rational man we see a creature given over entirely to the satisfaction of his lower desires.

With such wide fields opened up before us who will deny that at the moment the problems connected with the structure and function of the nervous system of man are among the most interesting by which the scientist is confronted. The ground, however, has as yet, only just been broken and the opportunity for work is enormous. In order that research along these lines may be effective a large corps of investigators with some leisure, and a certain amount of apparatus and well equipped laboratories are essential. There would seem to be something significant in the fact that just at the period in which these problems are being made accessible to investigation, here in America the conditions indispensable for properly attacking them promise to be provided. In a country where the tendency has been too prevalent in the past to prefer a smattering of knowledge of many kinds rather than skill and accurate knowledge in a narrower domain, we have at the present time evidences of a remarkable growth of the scientific spirit. We are coming to recognize that the man who

wishes to be *aliquis in omnibus, nullus in singulis* will in the end have an incomparably narrower outlook than he who slowly climbs the steep and difficult peaks of accurate investigation. With the ascent the latter is constantly attaining to an ever broadening view, until finally he reaches a position where there is opened up before him a panorama of truth wider than can be gained by following any other path.

While nothing is more to be deprecated than the total absorption of an investigator in fragments or the burying of himself in minute details, we in this country are in far less danger of stranding upon such a shoal than of shipwreck upon the sea of superficiality. Granted a thorough preliminary training, a mind of the first order runs no danger from engaging in special study. On the contrary the fruitful contemplation of great ideals first becomes possible when the man possesses some real living knowledge obtained directly from nature by his own exertions. No better example, perhaps, of this fact could be given than a references to the anatomical researches of Flechsig, some idea of which I have attempted to present to you in this communication.

THE STAGE SETTING OF THE NEUROLOGIST.

Dr. C. Fiessinger (*La Médecine Moderne*, Oct. 17th, 1896) in an article on psychical therapeutics describes the necessary moral and intellectual outfit of the successful neurologist: "He must indeed be a universally accomplished man, with the conviction of an apostle, the heart of a St. Vincent de Paul, the strength of a will of Napoleon, the patience of a confessor, the taste of an artist, the philosophic understanding of a Taine, the exquisite taste of a Parisian lady. All these the doctor must have, not to mention a solid learning and thorough education and the private virtues which serve as a basis for all the rest.

We have not mentioned as part of the psychical effect of the doctor on his patient, the stage-setting. Not that it represents an emotional factor which can be neglected, but only because the air of quackery about it is repugnant.

Charcot did not think so; theatrical in his clinic, he was still so at home. Patients coming to consult him did not pass at once from his waiting-room into the office; entrance to that sanctuary was not made with ease; a previous initiation must be gone through. This took place in a small, dark room, beautifully furnished, filled with elegant trifles and but feebly lighted. There he waited a quarter of an hour or twenty minutes, time enough to think about himself in the dark. All at once a ray of light penetrated it, a large door began to open, erect upon the threshold, bathed in a dazzling illumination, the God Charcot awaited him."

MITCHELL.

A CASE OF FRACTURE OF THE FIFTH CERVICAL VERTEBRA, IN WHICH AN OPERATION WAS DONE. DEATH ON THE EIGHTH DAY AFTER THE OPERATION.¹

By W. H. HUDSON, M.D.

Lafayette, Ala,

B. L. WHITE, age nineteen years, had been strong and healthy. He was in bathing in a shallow pond and jumped from a spring-board head foremost into the water; his head struck the ground and he fell to the bottom. Remaining longer than usual under water, his companions became alarmed and went to his rescue. He was paralyzed from the neck down, with the exception of slight motion in the shoulders and elbows.

I saw him first three weeks after the injury. He had been treated by electricity and drugs and an operation had been discussed with the patient and his family.

He was brought to me, a distance of fifteen miles, in an express wagon, and placed under my care for an operation, if it offered any hope of relief.

I found a fairly well-nourished, muscular young man, lying in bed with his head held slightly backward, palpebral fissures somewhat smaller than natural, pupils slightly contracted and not active to light; the arms abducted and rotated outward, the elbows flexed, the forearms pronated, with fingers falling over deltoid regions. In the left arm there was paralysis of all the muscles except the biceps, brachialis anticus, supinator longus and deltoid. In the right arm there was some motion still in the extensors of the wrist and elbow, with some power of pronation of the forearm, this, however, did not affect the characteristic position of the arms, the right and left maintaining the same position. All forms of sensation were absent below the second rib in front and the second dorsal spine behind. (See Fig. I.).

¹Read before the Chambers County Medical Society, Sep., 1893.

There was no demonstrable zone of hyperalgesia above the line at which sensation began, although the patient complained when sharply touched at any point about the face and neck.

His breathing was entirely diaphragmatic and varied

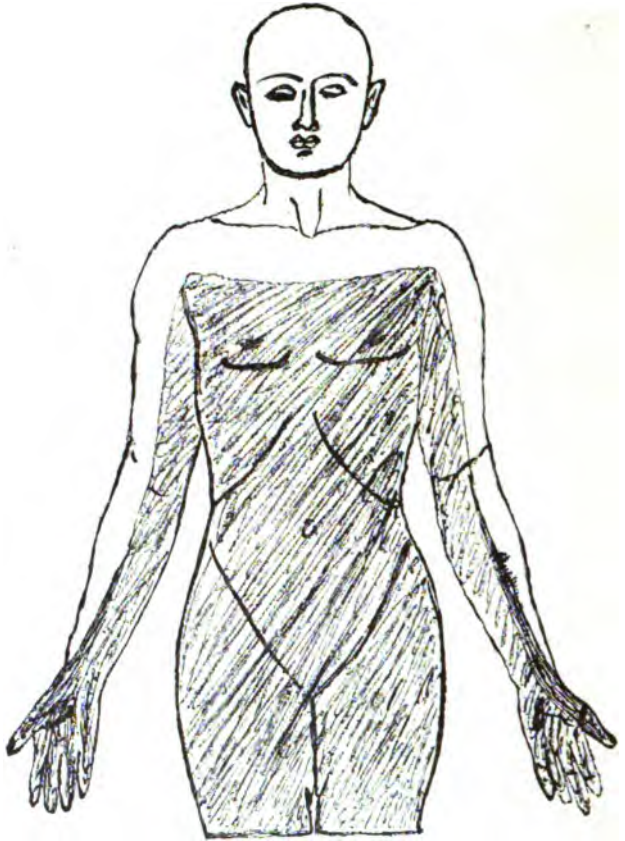


FIG. I.

between twenty and twenty-six respirations per minute; his pulse was regular and strong, though increased to about ninety-five per minute. His skin, over the paralyzed area, was dry, glossy and felt warm; there had been no perceptible perspiration over this area since the accident. Above the line of paralysis the skin was reddish and, more

specially over the face and neck, the perspiration was constant and sometimes well marked. There was a large bed-sore extending from the sacrum to near the tip of the coccyx, and further out on either side were other sores, somewhat larger but not so deep; there was yet another under the right shoulder which extended from the third dorsal spine to the angle of the right scapula; there were also reddened patches over both outer malleoli; below the umbilicus, near the median line, were four peculiar abscesses, each presenting a slightly rounded contour about the size of a silver dollar; the skin over them was not reddened, but in the centre of each was a small scab which was easily removed, leaving a round hole through the skin which had the appearance of having been cut out with a shoemaker's punch. Each of these abscesses contained



FIG. II.

from two to three drachms of pus and, after drainage and antiseptic cleaning, healed readily.

The bowels were moved mostly by enemata, although at times there was incontinence of fæces. The abdomen was generally distended, and flatus was passed at all times unconsciously. There was no priapism, but the penis was turgid and a glairy mucus was present at the meatus. There was incontinence of urine, and it dribbled away more or less constantly. The urine was highly ammoniacal, and there was a considerable degree of cystitis.

All the deep and superficial reflexes were totally absent.

When inserting the catheter to wash the bladder, no contraction of the muscles was found. Frequent careful examination with the finger of the anal sphincter showed

a total absence of contraction in that muscle. This is the first case recorded, of which I have any knowledge, in which this observation has been made. Examination of the spine posteriorly, and of the neck and throat, showed no abnormality. The temperature, which had varied from just a little above normal to 102, soon became more fixed, remaining most of the time less than 100. At two different periods his temperature rapidly rose to 104; but an enema reduced the fever each time, almost instantly.

The patient's appetite was good; his strength at times seemingly well preserved, at others appearing to decline.

He and his family were very anxious for an operation, if it offered any hope whatsoever.

Everything pointed to a total transverse destruction of the spinal cord, with probable degeneration from myelitis; but with all the facts placed before the patient, he yet urged an operation.

I decided, with very little hope of accomplishing any good, to operate as soon as the bed sore over the upper dorsal region had sufficiently healed.

The characteristic position of the arms and the limit of anæsthesia made it plain that the fifth cervical nerves were still intact; and that also on the right side there were fibres of the sixth motor nerve which were not destroyed.

Five weeks after the injury the operation was performed. I had decided to remove the laminae of the fifth and sixth cervical vertebræ.

All antiseptic and aseptic methods were strictly adhered to; the patient was placed on the operating table with a pillow under the abdomen and with the shoulders turned at an angle of about 45° and firmly held there by an assistant. Ether was given with satisfactory result; an incision was made six inches long over the vertebral spines, extending at the bones from the fourth cervical to the first dorsal. The tissues were cut rapidly away from the spinous processes and laminae of one side, the wound being packed with hot sponges while the other side was being operated upon. Hot sponges controlled the hemorrhage on this side; the sponges on both sides were held under the retractors while the canal was being opened. The hemorrhage throughout the operation was very satisfactorily controlled.

The spinous processes from the seventh and sixth cer-

cervical vertebræ were removed close to the laminae with curved bone forceps, and the lamina of the sixth cervical was removed with a rongeur. The dura-mater was slightly reddened and did not pulsate. There was no hemorrhage in the spinal canal nor within the dura. A curved probe was introduced under the lamina of the fifth cervical vertebra and revealed nothing abnormal, but when the body of the fifth was examined it was found to project backward and closed almost completely the spinal canal. See Fig. II.

The dura mater within this vertebra was lacerated on both sides at the point of exit of the nerves.

The spinal cord had unquestionably sustained a total transverse lesion and the operation was abandoned at this point.

The wound was united to its depths with silk worm gut sutures, and a small drainage was placed to the bottom and a careful protective dressing applied.

The operation lasted about one hour and produced no marked effect upon the patient's condition; hypodermic injections of strychnine had been given before and during the operation. After the effects of the anæsthetic had passed off the patient appeared little changed from the condition before the operation. He grew gradually weaker, however, and on the fourth day after the operation became delirious, and died on the eighth day.

His breathing was carefully noted and at no time did it assume the Cheyne-Stoke's character.

An examination of the wound only was allowed after death, and about six inches of the cord and the fractured vertebræ were removed. The drawing, Fig. II., was made from the fractured vertebræ with the assistance of the cut in Gray's Anatomy.

In jumping into the water, the patient's head must have been thrown violently forward, the fourth vertebra fracturing and forcing the body of the fifth backward into the spinal canal. The dura mater of the portion of the spinal cord removed showed a laceration about half an inch long on both sides. Further than this, it presented no marked change.

The spinal cord was firm at the lower part, but softer towards the injured portion and at the upper part; the upper portion appeared somewhat swollen. The crushed

part was very soft, being held together by the membranes. It was hardened in Müller's fluid and stained mostly with osmic acid.

The crushed portion was carefully examined in longitudinal sections and no nerve fibre found intact.

It is not to the credit of surgery to operate on any case when there is no chance for improvement.

In spinal injuries, the great question is, when should we operate and when should we not? It is clear that the operation in itself is not a dangerous one, and the danger of the operation should not be held, therefore, as a bar against its performance.

One thing is very evident, that injuries involving the cauda-equina are those which are most favorable for operation, and it is also evident that these are the cases in which delays are longest permissible. But as we approach the cervical region the injuries become more serious and operative relief less encouraging. What shall we do when the knee-jerks are abolished? We are led to believe that this indicates a total transverse lesion of the spinal cord, and in such a case, if any operation whatever should be done, it should be as soon as possible after the injury, in the hope that as many nerve fibres as possible may be saved from the inflammatory destruction produced by the pressure.

In those cases in which the reflexes yet exist we may wait (but I doubt the wisdom of such a course) the six or ten weeks which have been advised.

But in those cases *in which the reflexes are absent*, the earlier an operation is done the better.

STRONTIUM BROMIDE IN EPILEPSY.

Roche (Lancet, September 26, 1896) warmly commends bromide of strontium and reports in detail or summary 16 cases. All had been previously treated with other bromides and all showed better results from the strontium. In some instances the improvement was not marked, in others very striking. None could be considered cured, but 8 of them at the time of the report had been free from fits for periods of from 4 to 16 months. The ordinary plan was to give 20 grains of bromide of strontium and 5 to 10 grains of one of the other bromides three times a day. The strontium salt was increased to 1 dram when necessary to control the fits and acne was prevented by the addition of arsenic.

PATRICK (Chicago).

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Vice-President, Dr. Charles W. Burr, in the chair.

January 25th, 1897.

THE SENSE AREAS AND ASSOCIATION CENTRES IN THE BRAIN AS DESCRIBED BY FLECHSIG.

Dr. L. F. Barker, of Johns Hopkins University, Baltimore, read a paper with this title (see this Journal, page 325).

DISCUSSION.

Dr. William Osler, of Baltimore: One interesting point to me in connection with this subject is that twenty years after his early important contributions Flechsig should come forward with work so thorough and so satisfactory, still based upon embryological studies. In 1876, when Flechsig's papers appeared, I translated them with a great deal of industry, and they served me for many years a very important purpose. It is remarkable that this field should have been left practically unexplored all these years, except by Flechsig and his students.

Another very interesting point is that these observations, made from the developmental standpoint, should tally so accurately with those which have been made by experimental physiologists and clinicians. The accurate correspondence of the areas which Flechsig has worked out, with those which have been discovered experimentally and by clinical and pathological research, gives a very strong guarantee of the correctness of his studies.

Dr. Charles K. Mills: The Society is under great obligations to Dr. Barker for his admirable paper. The views of Flechsig are not widely known in this country, and, so far as I know, have never before been so carefully and thoroughly presented to an American audience. If accepted, they will compel us to revise the views on the subject of cerebral localization usually held in this country. While this is true, Flechsig's statements are, perhaps, not so revolutionary as they at first sight appear. His views, for instance, in regard to the functions of association areas in the prefrontal and midtemporal cortex are in practical accord with those held by many clinicians and physiologists. The part in cerebral activity assigned by Flechsig to his posterior, or parietal, association area is certainly different from that which is accorded to this region

by the majority of observers. I am certainly not in accord with his views regarding the angular, or angulo-occipital, region, believing it to be an area of higher visual representation, as held by Ferrier and others, and, as is well known, I am also not in accord with the views presented regarding the so-called sensori-motor area of the cortex, but I do not believe that it would be well to go into a discussion of this question this evening. One special point in the paper particularly interested me, and that was the statement of Flechsig's views as to the cortical terminus of the entering auditory tract, which, as I understood the speaker, Flechsig places in the convolutions caudad of the insula and within the Sylvian fossa—which are sometimes termed the retroinsular convolutions. In November, 1891, in the *University Medical Magazine*, and later in *Brain*, is reported a case of lesions of the superior temporal convolution. In this case word deafness followed a first apoplectic seizure, and complete deafness, with partial left-sided paralysis, a second apoplectic seizure some years later. An autopsy showed lesions of the first and second temporal convolutions of both hemispheres. The posterior two-thirds, or three-fourths, of the first temporal convolution in this case had shrunk to a thin slip. The retroinsular convolutions were two in number, and the posterior was very small and continuous with the posterior, much-attenuated half of the first temporal convolution.

Dr. William G. Spiller: Dr. Barker's paper is the first, I believe, in the English language in which a clear and lengthy presentation of this subject is given. The German journals have contained many references to Flechsig's views, and the criticism has not been altogether favorable. Some of Flechsig's statements are opposed to the observations of careful investigators. Men of the type of v. Monakow and Dejerine may not be called superficial, and the views which they have advanced after a study of cases for many years may not be put aside without consideration. H. Sachs has raised objections which have not been satisfactorily answered. It is well known that at birth the spinal cord is not fully developed, and that there are few medullated fibres within the brain. Flechsig has based his statements chiefly on the findings in the brains of very young infants at a certain stage of development. The author of the introduction to the new German journal, *Die Monatsschrift für Psychiatrie und Neurologie*, who, presumably, is Wernicke, calls attention to the fact that the brain of an adult is much larger than that of a new-born child. What is the origin and termination of these fibres of later development? Are we to believe that no more projection fibres are formed after a certain period in early life? Sachs' statement, that the intricacy caused by the gradual development of many fibres within the brain makes an examination by Flechsig's

method impossible after a certain age, is one of great force. We may accept the results of Flechsig's investigations as representing the condition of the brain at a certain stage, and as such they are of great value, but we would like more proof than has been offered that these relations remain unaltered in the adult.

If there are two areas which may be called association centres, it seems to me that these are the external part of the occipital lobe, with possibly a portion of the parietal, and the island of Reil, though even this is contrary to the statements of v. Monakow. There is a well-known band of fibres which connects the occipital and temporal lobes, and passes in part into the external capsule. Flechsig, I believe, has views in regard to the course of the inferior longitudinal fasciculus different from those held by most writers.

If I understand correctly the statements in regard to the cortical auditory centre, there are, according to Flechsig, fibres which pass from the first temporal gyrus into the external bundle of the peduncle. None of these arise in the second and third temporal gyri. Most investigators have held that this bundle comes from the occipital and temporal lobes. Dejerine and Vialet have shown that the occipital lobe does not give origin to such fibres, and the former, after an examination of six cases, believes that this lateral bundle is formed of fibres from the temporal lobe, chiefly from the second and third convolutions. Dr. Mills and I have studied and reported a case of abscess in the first temporal gyrus, extending very slightly into the uppermost part of the second temporal. There had been positive symptoms for sixty-eight days previous to death, though probably the lesion had existed longer. There was no degeneration in the lateral bundle of the peduncle by the method of Marchi. It seems to us that the case proves that fibres of the lateral bundle do not arise in the first temporal gyrus.

I do not understand the statement regarding the absence of projection fibres in the anterior association centre of Flechsig. It is very probable that this is chiefly an association zone, but if entirely so, in what part of the brain do the fibres of the anterior limb of the internal capsule arise? This is a question which has been asked by others. I present to the Society a few slides from a brain I am now studying. The area nourished by the middle cerebral artery is destroyed. It does not seem possible that fibres pass directly into the anterior limb of the internal capsule posterior to a perpendicular line drawn through the anterior part of the foot of the ascending frontal convolution, as the sclerosis has destroyed the Sylvian area back of this, and yet the anterior limb of the capsule is intact. The case is also in evidence of the correctness of the opinion, already mentioned, held by Dr. Mills and myself. The first temporal gyrus is destroyed, and yet there is no degeneration

in the lateral bundle of the peduncle after a lesion which had lasted many years.

Dr. L. F. Barker, of Baltimore: I have been much pleased with the interest which has been evinced by the members of the Society in this subject. The case of double lesion of the temporal lobe in both hemispheres, which Dr. Mills has reported is of great importance. Clinical and pathological evidence shows beyond doubt that destruction of both temporal lobes in the regions referred to, namely, the third-fifth and the fourth-fifth of the superior temporal gyrus and the transverse temporal gyri (or convolutions of the retroinsular region) leads to complete deafness.

I have been especially struck by the remarks of Dr. Spiller, inasmuch as some of the objections he has spoken of are very forcible, and their validity has been recognized by Flechsig himself. A number of the criticisms which have been made by the German neurologists are not, however, so damaging as one might at first think. While they cannot all be satisfactorily answered, yet a number of them have received at least a partial explanation.

In regard to the objection of Sachs concerning the possibility of further medullation after the period when the medullation of the cerebral white matter has become so diffuse that it is impossible to make out the appearance of new bands of fibres, I pointed out in my paper that Flechsig admits that a certain number of fibres may become medullated afterwards, although he does not believe that their number is very great.

I have done wrong if I have left the impression that Flechsig has studied only the brains of the newly born; on the contrary, he has followed up the subject, one might almost say, through every different stage of development, from the sixth month of fetal life to many months after birth, making sections through the whole hemisphere in different instances in various directions. Certain it is that in a babe three months old the sense centres are limited as definitely as he states in his report. It was in the spring of 1894 that Flechsig, while engaged in the study of a series of sagittal sections through the brain of a child three months old, first came to recognize the general principles underlying the development of the white matter of the cerebrum.

From the results of past investigations we have been led to believe that from the formation of definite areas in embryonic development we can draw valuable deductions with regard to function. We must, at any rate from Flechsig's studies, conclude that these sense centres are the principal end stations in the cortex of the various sensory conduction paths. If a certain number of fibres do become medullated later it remains to be determined in how far they really belong to the primary sense centres, and in how far they represent secondary conduc-

ions. It may be that the new method of studying the process of medullation, that of Ambronn and Held, in which the polarization microscope is employed, will aid us in settling these problems.

Hitzig, who believes that by the study of secondary degenerations it has been proven that there are some projection fibres belonging to the area designated by Flechsig as the "anterior association centre," admits that there are in this region very few such fibres.

The differences between von Monakow's results and his own have been recently discussed at some length by Flechsig in foot note No. 23 to his published address, *Die Localisation der geistigen Vorgänge*, and to this I would refer any who are interested.

Flechsig's statement regarding the fasciculus longitudinalis inferior of Burdach is, of course, in direct opposition to the views of Dejerine and others who believe it to be a band of association fibres connecting the occipital with the temporal lobe. The evidence that Flechsig brings forward as to the nature of the fibres concerned is suggestive. He says that, at an early period of development, the fibres of the so-called inferior longitudinal fasciculus are medullated before others in their neighborhood, and that they correspond to definite projection fibres of the occipital cortex, namely, to a portion of the optic radiation of Gratiolet. He thinks, further, that certain of the projection fibres run to the uncus; while others, running to the auditory sense area, have been supposed to be a part of this inferior longitudinal fasciculus; whence has arisen the impression that the fibres from this band are distributed to the uncinate gyrus and to the temporal gyrus. Flechsig's views upon this bundle are published in a special note in the *Neurologisches Centralblatt* about a year ago.

The case of abscess of the superior temporal gyrus, which Dr. Mills and Dr. Spiller have reported, with entire absence of degeneration of the lateral bundle of the foot of the peduncle, is of the deepest interest. If no fibres run from the first temporal gyrus into the base of the peduncle, Flechsig's view regarding the cortical origin of the lateral bundle of the base of the peduncle, his *temporale Grosshirnrinden-Brückenbahn* must be modified. In a note appended to his *Gehirn und Seele*, Flechsig states that he cannot define exactly its cortical origin, but that the fibres for the most part can be followed into the first temporal gyrus.

Dr. Spiller further suggests that it seems difficult to believe that the region called by Flechsig the "anterior association centre" is without projection fibres, inasmuch as the anterior limb of the internal capsule is not accounted for. I should have dwelt more at length upon this topic in my review of Flechsig's work, inasmuch as he has offered an explanation of the

difficulty. According to his findings in developing brains, several systems of projection fibres run through the anterior limb of the internal capsule, but all are distributed to regions of the cortex behind what he calls his "anterior association centre." Thus he finds in the anterior limb (1) projection fibres running to the frontal olfactory area and to the basal part of the gyrus fornicatus; (2) projection fibres passing to the feet of all the frontal gyri; (3) fibres running to the posterior end of the anterior third and to the whole of the middle third of the gyrus fornicatus, and, finally, (4) the fibres of the frontal cerebro-corticopontal path (Arnold's bundle in the base of the peduncle, Flechsig's *frontale Grosshirnrinden-Brückenbahn*). Curiously enough, some of the projection fibres mentioned, especially those distributed to the foot of the superior frontal gyrus and to the middle third of the gyrus fornicatus, run forward for a long distance, as shown in the diagram, in the white matter of the frontal lobe and arrive within two or three centimetres of the frontal pole. They do not, however, enter into cortical relations in this region, but turn at an acute angle to be distributed in the cortical areas referred to. Cases in which there has been disease of the front part of the frontal lobes, with resulting degeneration of the projection fibres, Flechsig would explain by an involvement of these subcortical projection fibres. He calls the attention of pathologists especially to the fact that in the fore part of the frontal lobe these fibres may easily be affected, and so lead the observer to faulty conclusions.

As I have already said in my paper, there can be but little doubt that a number of the ideas now being promulgated by Flechsig will scarcely stand the test of time. His anatomical studies are, however, of the greatest value, and will prove an immense stimulus to further investigations in the same field. If any one has the right to speculate, it surely is a man who has made as many positive unassailable contributions to our knowledge as has Flechsig. And should he in the field of speculation advance hypotheses which are not well founded we need not fear but that subsequent investigations will soon prove their falsity. Indeed, brilliant speculation even when in error is often a most important spur to accurate investigation. In deciding as to the most important contributions which Flechsig has recently made, all will probably agree with Prof. His of Leipzig, who suggests that the most stress is to be laid (1) upon the bringing of the anatomical proof of the existence of primary sense centres in the cerebral cortex and of the connection of these centres with the nervous apparatus situated lower down; and (2) upon the determination of the *successive* medullation of the single cortical areas and the exact periods of such medullation.

Periscope.

With the Assistance of the Following Collaborators:

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ANATOMY AND PHYSIOLOGY.

THE ELECTRICAL RESISTANCE OF THE HUMAN BODY. By Prof. Spehl and Dr. Sano. *Journal de Neurologie et d'Hypnologie*, No. 4.

By means of an ingenious apparatus, which gave very uniform, reliable results, the authors measured the resistance of the body to the galvanic current. The average found for healthy individuals was 1340 ohms. A slight increase in the resistance was found in diseases generally, but particularly in patients affected with nervous diseases. In one and the same disease great variations were noted in tubes for instance from 1600 to 4073 ohms. The authors find that according to their figures no constant relation exists between a given disease and the resistance stated in it, that accordingly *the electrical resistance cannot be used as a criterion for making a precise diagnosis of a nervous disease*. They deny that the electrical resistance is diminished in Graves' disease, noting even a slight increase instead.

Very marked and the most constant variations were shown to be produced by variations of the percentage of sodium chloride in the system. In one of the experimenter's, whose average resistance is 1450 ohms, this resistance went down to 1120 after the ingestion of 4 grammes of sodium chloride.

Sodium chloride solutions of from 5 pro mille to 10 pro mille concentrations were more sensitive in that regard than solutions of the strength of from 5 to 10 per cent.; that is, a slight variation of the former concentration modified the resistance in much higher degree, than variations of the latter percentage. It is interesting to note in this regard, that the average concentration of the sodium chloride in the human economy—5-10 pro mille—coincides with that concentration which is especially sensitive to increase or diminution of the electrical resistance by slight variations in the amount of the salt. This factor, the authors say, plays such an important part, that it may perhaps even be the principal cause of the variations of the resistance in diseases. In tabes, for instance, the crisis gastriques may so alter the nutrition, and consequently the chemical composition of the tissues, that this alone might explain the high figures (4073 ohms, etc.) found in some tabetics.

ONUF.

A STUDY OF PAIN.

H. Meige gives an abstract from Charles Richet's "Etude Biologique sur la Douleur" (*Journal de Neurologie et d'Hypnologie*, 1896), of

which the following is a summary: Pain is produced by strong excitations. All abnormal states provoke pain. The persistent memory of a painful excitation is one of the fundamental characteristics of pain. Pain is in relation with the finality of beings; this is the only psychological preventative defense. Living beings divide themselves into two groups as to preventative defense against dangers. Some fortify themselves by instinct; they have not painful sensations, and they are not able to modify their acts in the face of the unexpected. Others fortify themselves by intelligence. They have the comprehension of pain and have no wish to expose themselves to it. To suppress pain and its recollection would be to reduce human consciousness almost to nothingness.

MITCHELL.

PARALYTIC INFLUENCE OF ATROPINE UPON THE GLUCOSECRETORY NERVES OF THE LIVER.

Cavazzini and Soldaini conclude their experimental researches as follows: That atropine exerts a paralyzing action upon the nerves of the coeliac plexus; that these regulate the production of sugar in the liver; that the production of glucose must be regarded as a true secretion, and the corresponding nerve fibres must be regarded as the true glyco-secretory fibres.

KRAUSS.

NEURO-PATHOLOGY.

TWO CASES OF CONGENITAL, SPASTIC RIGIDITY (LITTLE'S DISEASE) WITH AUTOPSY. *Comptes Rendus Hebdomadaires des Séances de la Société de Biologie*, March 31, 1897. By J. Dejerine.

Case 1. The patient had been born at term and had been paretic in the lower extremities since early infancy. When examined at the age of seventy-nine he presented spastic paraplegia of moderate intensity without atrophy, and was able to walk with a cane. The patellar reflex was exaggerated. The upper limbs and face were not affected. Sensation and the action of the sphincters were normal. Intelligence was of the average degree. Since infancy the patient had been subject to epileptic attacks. There was a cavity in each hemisphere which communicated with the lateral ventricle; the one on the right side was situated at the junction of the middle and lower thirds of the fissure of Rolando, while the one on the left was at the junction of the superior and middle thirds of the corresponding fissure. The convolutions converged toward the crateriform depressions. There was no degeneration of the cerebral peduncles or any part of the oblongata or cord. In the lateral columns of the cord and in the pyramids there was an overgrowth of the neuroglia, which may have been due to the advanced age of the patient.

Case 2. The patient was born at term and was said to have presented rigidity at birth. The first years of life were passed in bed, but about the age of nine or ten the arms began to be less stiff, and the patient was able to walk with crutches. There were no epileptic attacks in this case. At the age of forty-four the intelligence was below the normal, and there was spastic paralysis of all four limbs, more marked in the lower, and especially on the left side. The left lower limb was smaller than the right. The patellar reflexes, especially the left, were exaggerated, and ankle clonus was observed on the left side. The power of movement was slight in the lower limbs, and walking was only possible with crutches. The man walked as if without joints (*tout d'une pièce*) in swinging the lower limbs. The arms could be moved more freely, but still very imperfectly, and the left was atrophied. The olecranon reflexes were exaggerated, and the contractures were greater in the left limb. Sensation and the sphincters were not affected.

There were no lesions of the cortex or central gray masses, though the cord was smaller than is normal in adults. The anterolateral columns of the cord were smaller, but the posterior appeared to be normal, as viewed by the microscope, except at one part to be mentioned. The left anterior horn was a little smaller than the right. The motor cells were equally numerous on the two sides and appeared as in normal cords. Between the first and second cervical segments there was a circumscribed lesion. The posterior horns at this level were destroyed and replaced by sclerotic tissue extending into the lateral columns and containing numerous vessels. There were also many thickened vessels in the posterior columns, but no sclerotic tissue. Ascending degeneration was not observed in the posterior columns, but the pyramidal tracts presented retrograde and descending degeneration.

Dejerine states that this second case demonstrates that the symptoms of Little's disease may be due to a primary, focal, spinal lesion, which has developed during the intrauterine period, and is possibly the result of an infectious process. The sclerosis and agenesis of the crossed pyramidal tracts were probably due to this focal lesion. The explanation for the predominance of the symptoms in the lower limbs is not easily found.

SPILLER.

MOTOR AND SENSORY APHASIA.

Mantle (British Med. Jour., Feb. 6, 1897) reports two cases of aphasia that seem to merit rather complete notice. The first especially is interesting and instructive.

The patient was an artist; there was a suspicion of syphilis, but no positive evidence of it.

At the age of 31 he suffered from several slight epileptic convulsions, and after one of them was paralyzed on the right side and was aphasic, but recovered completely in 14 weeks except that some slight weakness of the leg remained. A year later he had a similar but less pronounced attack. He completely recovered from this and went back to work, but suffered from headache. At the age of 41 he awoke one morning perfectly conscious, but speechless. Examination the same evening disclosed complete motor aphasia with slight weakness of the right side of the body. He could write correctly, but was emotional and easily excited. He gradually became drowsy and the hemiplegia became complete. There was gradual improvement up to the 11th week, when in the course of three days he became totally deaf, blind and dysphagic; the pupils reacted to light and the optic fundi were normal. He remained deaf and blind for ten days, but could recognize his wife and the nurse by touch. He gradually regained sight and hearing, and what is peculiar, could at first recognize sounds at a distance (the barking of a dog, a piano in the adjoining house), but not near by. This fact was recognized by the patient and he inquired about it by writing on the coverlet with his finger. It was now found that he had right hemiopia, and could recognize whispered words, but not those spoken in a loud voice. After ten days of improvement he again became entirely blind and deaf, and communicated with others like a blind, uneducated deaf-mute, but it is worthy of remark that he retained the conception of color and form; e. g., to express his wish for milk and soda he pointed to the sheet to indicate white and drew the form of a siphon in the air with his finger. To indicate a chop he drew the form of it in the same way, and likewise indicated that he wished some lilies of the valley placed in his hand. After a few days sight returned, although he remained hemiopic, he was able to speak somewhat, and motion in the extremities improved but they were very sensitive to touch. He now again understood a few whispered words. As hearing improved it was found to be

better on the right side than on the left, and the sense of smell was distinctly impaired on the left. Gradual improvement followed, but six months after the attack memory was almost nil, and after ten months he was still hemipic and speech was very poor. About ten and a half months after the attack he awoke from a nap with a dazed look, and was found to be hemiplegic. By the following morning sight was lost, hearing for words abolished, although it remained acute for sounds, and he had complete agraphia, but could communicate by means of signs. The blindness and word-deafness lasted four weeks. "While deaf and blind to words, as well as having complete paralysis of motor speech, he expressed a wish to make his will, and made his wishes known by forming the outline of the letters on the sheet of the bed. He also made his lawyer and wife understand that when they meant 'yes' they were to tap his hand, and when 'no' they were to cross it. To satisfy him the will was executed, and with his left hand he very badly wrote his signature."

He again gradually regained motion and his faculties, the motor part of speech being the slowest to improve, as he was unable to speak for thirty-two weeks after the beginning of this second part of the attack. Two years after the beginning of the trouble he could walk slowly with the aid of crutches, and soon after began to take great pleasure in sketching from nature, although his mind was a complete blank with regard to paintings he had done before his illness.

"It was very curious, how in the early period of his convalescence movement of the body, that is, to another room, brought on the sensory disturbance of speech for perhaps twenty-four hours. This occurred on three or four occasions."

The author continues: "Four years have now elapsed since the attack came on, and I have quite recently had a chat with the patient in his studio. He tells me he has tried hard to paint during the last year, but he is so disgusted with his work that he has practically given it up. To my uncritical eye his work looks very passable. He says, however, that his difficulty is in trying to remember how he used to do it, and when he finds he is doing the thing wrong, he is so dissatisfied with his efforts that he has ceased making further attempts. He can, however, do pen and ink work exceedingly well. On asking him if he remembered painting the pictures about his room, he said he did not, but still thinks he saw another man paint one of them. He described himself as being a better judge and more critical than ever he was before, but no workman; and this is not through want of muscular power, for that has quite recovered."

"He has a very good vocabulary, however, when talking, and never wants for a word, though there is sometimes an occasional hesitancy in pronunciation. Contrary to Lichtheim's experience, the sensory aphasia in this case of total aphasia cleared up before the motor."

Case 2 was a man undoubtedly syphilitic. Five years before there had occurred a sudden paralysis of the extensors of the left forearm from which he recovered, and two years and a half later he had epileptoid seizure with some weakness on the right side. He also recovered from this, but at intervals had slight fits. One of these, which was attended with twitching of the right face, ushered in the present trouble. On examination he was found to be language deaf, answering to all question: "I really cannot tell you, I am sure, just now." He could not read or recognize most objects. There was no paralysis and "the motor part of speech was good," but in the next paragraph the author says: "Some things shown he remembered easily and could pronounce their name, others he could not name; but if he thought for a time he sometimes gave the correct name or a similar sounding word, or substitute a roundabout phrase as an equivalent. Occasionally, when taken suddenly, he seemed to answer instinctively

and generally correctly. A pencil he knew the use of, but could not name. A pen he called 'pea,' then pen. A knife and fork and book he knew, but called a bottle 'brot,' and medicine he could only pronounce partially. This would seem to us to indicate that there was also some motor aphasia. He was agraphic to spontaneous effort, but could copy, although incorrectly. There was improvement for a few weeks, when he was found unconscious and after this passed off there was hemiplegia of the right side and complete aphasia. He again improved, but six weeks later had another seizure, followed by complete right hemiplegia and speechlessness. He was restless and noisy and removed from the hospital.

We may quote from the author's comments on the first case without endorsing them.

"The centre for motor speech (the posterior part of the third frontal convolution and the lower part of the ascending frontal on the left side) were first affected, and the ascending frontal convolution containing also the cortical centres for the movement of the tongue and lips accounts for the early loss of these movements in this case. The part of the brain concerned in the sense of hearing was next affected, which is the posterior half of the first temporo-sphenoidal convolution on the left side. When this region is diseased there is often transient deafness in the opposite ear, as was the case here. The fact observed that he could hear a whisper and understand the word when whispered very softly and slowly is one I find no record of, but points, I think, to over-stimulation of the centre by the ordinary voice, preventing perception of sound characters. The hearing of both ears went simultaneously, but the right, or that on the opposite side of the lesion, was the first to recover."

In a word blindness a lesion of the first temporal convolution is found in the majority of cases. The hemianopsia was probably due to involvement of the occipital lobe, for the pupil still continuing to respond to light supports Wernicke's suggestion that the symptom is often due to a lesion in the occipital cortex. The amaurosis which has been present in both attacks was probably cortical also. The optic nerve always looked healthy. Mind or object blindness was part of the usual disturbance. It has been produced artificially by Munk in dogs by extirpating parts of the occipital lobe. Loss of smell, which was associated with the sensory aphasia, is thought by Gowers to be due to softening of the root fibres at the commencement of the fissure of Sylvius. I have not mentioned the fact before, but this patient had visual hallucinations to which Prof. Henschen, in his work on Pathology of the Brain, directs attention, as likely to trouble a patient with homonymous hemianopsia when that symptom is of cortical origin. He said he could always see in the blind half of the field of vision a man at the back of his head holding two lighted candles. Bastrih has strongly pointed out that lesions in the posterior parts of the brain are more apt to produce hallucination than lesions in the middle or anterior parts. The amount of sensory disturbance was very remarkable in this case, and might have suggested at an early period a pure-functional origin; but as Wyllie says, in Disorders of Speech, "The very existence of word deafness or word blindness in any case should suggest an organic rather than a functional cause."

In the same journal Allan reports a most unusual case of complete aphasia.

A bricklayer, aged 34, after being a teetotaler for seven months and again been drinking for two weeks, when while walking on the street, he felt some dizziness and noticed a slight weakness of the left leg. On arriving at his home he discovered that he was unable to articulate a single word, although able to express himself freely in writing. On examination the following morning he complained of oc-

casual attacks of faintness, of a swimming sensation in the head, and of a tingling in left leg up to the knee.

The apex beat was outside the nipple-line and an aortic diastolic murmur was present. Intelligence, memory and attention were good. He understood all questions and answered them promptly and correctly in writing, but could not utter a word. He could understand what he read, copy words and figures, write spontaneously and from dictation.

Although his general condition improved and he resumed work, loss of speech remained absolute until nine weeks after its disappearance. While at work he fell a distance of six feet, landing on his head. He was somewhat stunned, but almost immediately called his companion by name, and from that time was able to talk. The author reaches no conclusion as to the nature of the case. PATRICK.

HEMIANOPSIA IN CEREBRAL ABSCESS OF OTIC ORIGIN. Lannois and Jaboulay. *Gaz. Med. de Paris*, 37, 1896.

The authors report a case of cerebral abscess due to extension from an otitis purulenta, which, according to their researches, is the ninth case in literature giving right-sided hemianopsia.

Further symptoms were agraphia, alexia, optic aphasia and right-sided facial paralysis. Upon autopsy there was found diffuse encephalitis of the central lobe of the left hemisphere and at the base of the frontal convolution; and in addition a large abscess involving the greater part of the central portion of the left occipital lobe. JELLIFFE.

EXPERIMENTS ON OPERATIVE EXPLORATION OF THE BRAIN. Payr. *Centralb. für Chirurgie*, No. 31, 1896.

To determine what could be learned by exploration through very narrow openings in the skull, the author performed a series of experiments upon dogs. He made use of drills 1 and 1½ to 2 mm. in diameter, with adjustable shields (to prevent sudden perforation and wounding of underlying structures), operating with all aseptic precautions. The questions which he attempted to decide were: 1. If it was possible with straight and curved exploring needles to reach collections of fluid beneath the membranes or in the brain substance. 2. If using little harpoons of special construction, particles of brain substance or membranes could be removed and examined microscopically. 3. If it could be determined whether the dura pulsated or not. For this latter purpose he used little glass tubes filled with sterilized water, and also closed tubes with end drawn fine, and bent at a right angle to serve as indicators. The five dogs operated upon recovered promptly, and seemed to have experienced no ill results. All the questions were decided affirmatively. To determine how far he could reach with the exploring needle, he injected through the drill-hole the brain of a dog—dead from another experiment—with alcohol, methylene blue solution, and found on section that the fluid was distributed over 10 to 12 cm. of surface, and penetrated 4 to 6 cm. At autopsy made three weeks after the operation, showed healing with adhesion between dura and bone, and with but a slight depression where the brain substance had been removed by the harpoon. The opening in the bone was filled with a firm reddish-yellow mass. He does not attempt to say how far this procedure is applicable to human beings, but suggests that it may be of use in locating hemorrhages, cysts, or tumors, and obtaining specimens for microscopic examination; also for tapping the lateral ventricles. C. L. ALLEN.

POLIOENCEPHALOMYELITIS.

The disease is most common in persons between 20 and 60 years of age. It is usually subacute in onset, rarely acute or insidious,

most progressing subacutely and later pursuing a chronic, stationary or varying course. Manifestations of constitutional disturbance, such as fever, headaches, vertigo, vomiting, are generally wanting throughout. In most cases the ocular muscles are first involved and in irregular order, one after the other. Ptosis may appear first on one side and then on the other and may be slight or pronounced. Then the other muscles become involved, usually in greater degree upon one side than upon the other. The fourth and sixth nerves are not spared. Diplopia is not always present; and almost never well defined strabismus. The internal ocular muscles usually escape. The disturbance of the ocular muscles generally recedes, to give place to bulbar or spinous symptoms, or they may remain in association with the latter sometimes the disease sets in with bulbar symptoms and rarely with weakness of the muscles of the neck, trunk and extremities, advancing upward. Usually extension occurs in stages, weakness in the upper or lower extremities being suddenly added to the paralysis of the ocular muscles; or paralysis of the muscles of deglutition or the ocular muscles being added to weakness of the neck. Not rarely bulbar symptoms predominate, including weakness, fatigue, permanent or progressive paralysis of the muscles of the face, tongue, those of mastication, of deglutition; those of the œsophagus in unequal degree upon both sides. In the extremities the parts nearest the trunk are involved earlier than those at a distance; the extensors at times more than the flexors.

The tendon-reflexes, and especially the knee-jerks, are enfeebled or lost. The sensory and the sensorial functions usually remain uninvolved, as well as psychic activity and the sphincters. Fibrillary twitching is not observed. Muscular atrophy does not take place as a rule; if at all, late and in slight degree. Paralysis is the primary and the most conspicuous manifestation. The electric reactions remain unchanged or are enfeebled and in grave cases, perhaps, lost. Degenerative reactions are rare. The so-called myosthenic reaction is observed in some cases—that is the muscles respond normally to faradic currents of ordinary intensity, but to tetanizing currents the response grows gradually more and more feeble until it ceases altogether. It appears most marked when the fatigue-phenomena or voluntary innervation are quick and pronounced.

The intensity of the palsy is variable. Fatigue and recovery take place readily and quickly. The degree of fatigue does not always correspond to the degree of permanent weakness and paresis. In severe cases the patients become completely helpless. In walking, the knees may give way; or the gait may be staggering. Active exercise may be attended with an urgent sense of the need of air, from fatigue of the respiratory muscles. Not rarely sudden death occurs from paralysis of the vagus or of the diaphragm. Other dangers may arise from sudden weakness of the muscles of the pharynx and œsophagus, so that solid diet may be necessary and regurgitation through the nares may take place. If the hands are involved there may be difficulty in carrying food to the mouth, in writing and in other manual exercises. When the eyes are used, the ptosis is aggravated and the muscles of the neck become weaker from efforts to hold the head erect. Speech becomes nasal and after a time indistinct, low and whispering. The ability to sing and to whistle is lost, and smoking becomes difficult.

A characteristic feature of the disease is the rapid recovery of strength after rest and the improvement in the morning after sleep. Remissions in the intensity of the symptoms occur from time to time and may continue for varying periods—from days even to years—and culminate in perfect recovery. The progress of the disease may further be very slow and characterized by exacerbations; or the morbid pro-

cess may finally cease to progress, leaving a condition of permanent weakness. Sometimes death occurs unexpectedly.

In many cases no lesion has been found after death. In one case degenerative changes were present in the medullary sheaths of the nerve-roots of the medulla oblongata. In another there was vascular dilatation, with hemorrhage and degenerative-atrophic conditions of the ganglion-cells of the central gray matter. These various lesions, however, are inconstant and inadequate to explain all of the symptoms; so that it must be assumed that there occur chemic, nutritive or toxic changes capable of disappearing and of repair without leaving evidences of their previous presence.

In an etiologic connection most of the cases reported have had some direct or indirect relation to one or another of the infectious diseases; others to over-exertion or to excessive use of alcohol or tobacco. No relation to syphilis could be established.

Subacute or chronic poliiencephalomyelitis is to be distinguished from acute encephalitis by the absence of acute onset, of constitutional manifestations (fever, delirium, etc.) and other cerebral symptoms (aphasia, etc.); by its peculiar and more systematic invasion (bulbo-spinal symptoms) and its chronic, partly remitting, partly progressive course. Poliiencephalomyelitis differs from acute poliiencephalitis in the absence of symptoms of general disturbance (vertigo, vomiting, optic neuritis, delirium, etc.), in the less rapid course and in the presence of bulbar and spinal symptoms. From progressive bulbar paralysis it differs in the less characteristic localization and a less universal involvement of the bulbar nuclei, in its irregular, fluctuating, remitting course, in the absence of fibrillary twitching and of degenerative reactions, in the relative absence of muscular atrophy, in the presence of muscular fatigue, in the possibility of improvement, in the absence of distinctive lesions, in the early and frequent involvement of the ocular muscles and also of the neck and extremities, and in the irregular, abrupt involvement of the entire system of bulbo-spinal nuclei. From amyotrophic lateral sclerosis it is to be differentiated by the absence of spastic manifestations and from progressive muscular atrophy by the varying course, characteristic localization, atypical distribution, unsteady progression, early participation or precedence of paralysis of bulbar nerves and of the ocular muscles. It is to be distinguished from pseudo-bulbar palsy by the mode of onset and by the presence of psychic disturbance and of hemiplegia.

In the treatment of poliiencephalomyelitis drugs have proved of no service. The best results will be secured through rest and the avoidance so far as possible of muscular activity; every unnecessary fatigue should be avoided. Solid food should be interdicted and only small quantities of soft food and of liquid given at frequent intervals. In aggravated cases the administration of nutritive enemata may answer a useful purpose. Especial attention should be directed to prophylaxis in so far as the danger of infection is always to be avoided, and should such effort prove futile the acquired disease, with its attendant intoxication, should be gotten rid of with all possible expedition. During convalescence from infectious diseases undue physical effort should be avoided and roborant treatment should be instituted.—*Journal of the American Medical Association*, Nov. 21, 1896.

H. L. S.

CORTICAL EMBOLISM IN THE INSANE.

Tomlinson and Chilgren (*Med. Record*, August 1, 1896) report a case of what they term "cortical embolus (red softening)." Their paper may be considered as a brilliant example of how not to report a case. Four-fifths of the contribution are devoted to an imperfect clinical report and irrelevant post-mortem details. No evidence is adduced of embolism of a single vessel and attention is called to no

source of the supposed embolus of emboli. There is no adequate evidence of softening, red or otherwise, and regarding the cerebral vessels about the only pertinent statement made is that "a thrombotic condition of the arteries and veins of the cortex existed throughout." There may be such an entity as "a thrombotic condition" it is entirely too uncertain and illusive in its nature as presented by the authors to be of any use to the reader. Finally there is some description of findings by means of the Golgi or probably a modified Golgi impregnation. To those conversant with the uncertainties and difficulties of this method in pathologic (and purely anatomic) research, this report of cell changes as well as the accompanying reproductions from photomicrographs are of little interest and less value.

PATRICK (Chicago).

LES MYELITIS INFECTIEUSES. By Dr. T. B. Crocy. *Journal de Neurologie et d'Hypnologie*, Nos. 1 and 3.

In tabulating all cases of myelitis reported in which the disease came as in connection with infectious disease, C. comes to the conclusion that by far the most cases have been observed in connection with diphtheria. Variola comes in the second order as to frequency; then comes intestinal affections, pneumonia, blenorragia, staphylococcism, typhoid fever, erysipelas, influenza, measles and finally as the least frequent cause articular rheumatism.

C. finds that the prognosis of these "infectious" myelitides is relatively less grave than that of the other myelitides.

Those cases in which no post mortem proof existed were not considered in the table. The cases observed in connection with scarlet fever and with malarial fevers were not included for this very reason.

Be it understood that under the title of myelitis C. includes (clinically speaking) infantile spinal palsy, the acute spinal palsy of the adult, the tabes dorsalis spasmodique, diffuse acute or chronic myelitis, etc.

ONUF.

Über Rückenmarkserkrankungen bei Keuchhusten. (Lesions of the Spinal Cord in Pertussis). Verein für innere Medizin, Berliner klinische Wochenschrift, No. 45. By Bernhardt.

Spinal lesions in whooping-cough are rare. Bernhardt reports the case of a child, who on the tenth day of the disease suddenly lost power over its lower limbs. There was neither fever, nor convulsions, nor disturbance of consciousness. The upper extremities were not affected. The tendon reflexes in the leg were exaggerated and sensation was less acute. The vesical functions were impaired. After a few years the child entirely recovered. The writer thinks the cause may have been hemorrhage into the spinal canal or cord, secondary infection, or intoxication from the products of the bacteria.

SPILLER.

EMOTIONAL DYSGRAPHIA.

M. Féré (*Médecine Moderne*, Nov. 18, 1896), at the Soc. de Neurologie, communicated his observation of a patient presenting the phenomenon of writers' cramp, if watched by anyone or writing from dictation. Under these conditions the hand became immovably fixed and the patient could not write a word. After a minute or two of effort he could write without difficulty and no trace of the disability in his handwriting. The same trouble was not experienced if the patient wrote in a room by himself. No statement is made as to whether this difficulty was accompanied by pain or not, and M. F. considers it in "no way analogous to writers' cramp." MITCHELL.

Book Reviews.

AN AMERICAN TEXT-BOOK OF PHYSIOLOGY. By H. P. Bowditch, M.D.; J. G. Curtis, M.D.; H. H. Donaldson, Ph.D.; W. H. Howell, Ph.D., M.D.; F. S. Lee, Ph.D.; W. P. Lombard, M.D.; G. Lusk, Ph.D.; W. T. Ponter, M.D.; E. T. Reichert, M.D.; and H. Sewall, Ph.D., M.D. Edited by William H. Howell, Ph.D. M.D. W. B. Saunders; Philadelphia, 1896.

The present portly, if not over-bulky tome, is an excellent representative of the class of works, filled with good material with too much padding. Fully one-quarter of its matter might have been pruned. One of the causes of redundancy is the overlapping of subjects; this is quite marked in the physio-chemical portions of the volume. There is, we think, a lack of proportion in the treatment of the different chapters; some, while very excellent in themselves, are too encyclopædic for a text book; while others are pushed for space events express a bare outline of the subject.

This later defect, we believe, is especially evident in the chapters upon the nervous system and the special senses.

In the chapter upon the nervous system the author presents much of the matter already published in his work upon the "Growth of the Brain," which, while very valuable as information, is, we consider, at times apart from the subject in hand. Much of the more recent anatomico-physiological knowledge of the nervous system is entirely omitted, and in the chapter upon the special senses the lack of this same element constitutes a glaring fault of omission.

From the standpoint of workmanship the book is fair, being well printed and illustrated; but, as already indicated, it is too bulky.

THE YEAR-BOOK OF TREATMENT FOR 1897. Lea Bros., Philadelphia.

This is the 13th issue of this "hardy annual." Its 700 pages of closely-printed text has been carefully prepared by well-known English writers. Dr. Ernest S. Reynolds has the Department of Mental and Nervous Diseases directly under his charge. Twenty pages have been devoted to his department, and deals with only seven subjects. The same may be said of this book as of the American Year-Book, namely, that not enough space and matter are devoted to this important department.

THE AMERICAN YEAR-BOOK OF MEDICINE AND SURGERY. 1897. W. B. Saunders, 925 Walnut St., Philadelphia.

The volume for 1896 of this admirable résumé of the recent advancement and researches in the various departments of the medical sciences received such universal recognition that it seemed there would hardly be much left for any one to note and advantageously arrange so as to be useful as an additional accession to one's library; but the nearly twelve hundred pages of matter that are given to us to examine teems with information for the student and investigator in every branch. Out of the sixteen departments, ten are arranged by Philadelphia contributors. The rest is divided among well-known men of New York, Chicago, and Cleveland, Ohio. That on nervous and

mental diseases is under the direction of Archibald Church, M.D., and Hugh T. Patrick, M.D. It is by no means as complete in its compilation and analysis as other departments in this work. If the editors consider that 69 pages is sufficient to review the work in this department of medicine, or if there has been a feeling on the part of the contributors that they have exhausted the subject, we can only say that neurology has not been given a fair representation. The arrangement of the subjects in hodge-podge confusion is to be deplored. What is noticed, however, is fairly well and concisely abstracted, but only found by the most excellent of indexes. The work is well illustrated and the printer's work most excellent; and it will prove a ready practical work. It is not a question any more, why systems of medicines and encyclopedias are being superceded by these yearly resume works—the question is one of up-to-date versus out-of-date; and a practical work of this kind should be as complete as possible all along the line of the recent advances.

A TEXT-BOOK OF MATERIA MEDICA, THERAPEUTICS AND PHARMACOLOGY. By Geo. Frank Butler, Ph.G., M.D. W.B. Saunders, Philadelphia.

The excuse to write another text book, in this case, is the most valid one, if the finished work is of any value, and the apology lies in Dr. Butler, being a professor of materia medica, desires to use in teaching what he considers a clear, concise and practical work. When one begins to test an efficient remedy, he starts on the road labeled "faith," and begins to reflect; so when a book is labeled for permanent reference, no less than the requirements of the class room. The sign board speaks with an authoritative tone that challenges investigation. The first thing the reviewer did, was to ascertain about the treatment of vertigo. He found a most admirable index and found cubeb, page 351, and valerian, 388. As to reference to inform him, "Internally—cubeb is recommended in certain nervous disorders, such as headache, impaired memory, vertigo, and fainting, and has been thought beneficial in certain cases of paralysis;" and valerian, "Nervous headache and vertigo due to cerebral anæmia and irregular distribution of blood are, in the majority of cases, promptly relieved by valerian or ammonium valerianate." As this comprises the treatment of vertigo, we feel that in this book, at least, we have not received our money's worth; disappointment seems to start with the investigating traveler at the outset. As far as the neurological therapeutics in the entire work go, it is only fair, there is the old fault of, "it is reported to be of value in—." We like to see therapeutics obtain a positive purgation of other authentic opinions and positive assertions, and give the readers the impression that at least the author had a confidence in his opinions and written statements, and had thoroughly tested doubtful inferences. The description of drugs and the toxicological consideration is excellent. He distinguishes between an "unlowered action" and "poisonings;" by the former he includes the susceptibility and idiosyncrasy of the patient toward certain drugs, and the later refers directly to definite influence. The chemical and pharmacological departments are treated sparingly throughout the work. There is, however, a very valuable reference chapter, on page 49, on pharmaceutical preparations, and extends 75 pages. The classification then follows of considering the drugs under disease medicines, antiseptics, symptom medicines, and topical remedies. Our popular publisher, W. B. Saunders, has made a handsome, well-printed, agreeable-sized and reasonable book, and we prophesy that if the author's students peruse and con well the contents, that they will be fairly equipped to all dosing requirements of modern and ancient times.

ANATOMY, DESCRIPTIVE AND SURGICAL. By Henry Gray, F.R.S. Lea Bros. & Co.

Gray's anatomy needs no introduction nor word of commendation. The present, 14th edition, shows many new plates, one hundred and fifty-five, the publisher says. There are also new and re-written chapters, especially is this noted in the section devoted to the brain, which has been not only entirely remodelled but re-written by Dr. Bern B. Gallaudet, Demonstrator of Anatomy of the College of Physicians and Surgeons, while one might cavil with the author in many ways, yet one must confess that his work is well done, and applaud him for the fact that he has stuck to English nomenclature. The entire work shows improvement and impresses one that it is destined to enjoy still longer its exalted position.

ESSENTIALS OF PHYSICAL DIAGNOSIS OF THORAX. By Arthur M. Corwin, A.M., M.D. W. B. Saunders, Philadelphia.

A most excellent little work. It brightens the memory of the differential diagnostic signs, and it arranges orderly and in sequences the various objective phenomena to logical solution of a careful diagnosis.

PRACTICAL POINTS IN NURSING FOR NURSES IN PRIVATE PRACTICE. With an appendix. By Emily A. M. Stoney.

This is rather an extensive work on all kinds of first aid to the injured, bandaging, dose lists, and glossary of odd and end information for the nurse's benefit, profusely illustrated and beautifully arranged and bound by the ever more and more popular publisher, W. B. Saunders, of Philadelphia.

A MANUAL OF OBSTETRICS. By W. A. Newman Dorland, A.M., M.D.

It is a work of over 700 pages, and is a compendium of the best work and ideas of the day in excellent arrangement and in good and concise English. While not being a startlingly original work, its tone is firm and implies a confidence, and it is an excellent guide for the student.

SWEDISH MOVEMENTS, OR MEDICAL GYMNASTICS. By Dr. T. J. Hartelius, Director of the Central Gymnastic Institute of Stockholm, Sweden. Translated by A. B. Olsen, M.D. With introduction and notes by J. H. Kellogg, M.D. Published by the Modern Medicine Pub. Co., Battle Creek, Mich. 1896.

In the present translation of Dr. T. E. Hartelius' work, first published in the original in 1884, on Swedish movements, the reader will find an excellent text book on that branch of Swedish gymnastics, which deals with the treatment of deformities and disease, a subject which, in a superficial sense, has long been known to most of our readers, but which, until now, has never been presented to them in a scientific essay. The work itself is admirably written, giving a clear description of the entire system of Swedish gymnastics with its characteristic features of graded exercises (passive, free and resistive). It is shown how these exercises are influenced by position; and how, by means of suitable support to certain parts of the body, they can be limited to any special part or set of muscles, their action thereby becoming definite and distinct. The physiological effects of the various movements locally, or on the body as a whole, are considered in detail; making it plain how, under scientific management, these movements can in a variety of ways be made to affect certain pathological conditions most beneficially. What may strike the American reader as curious is to find what he knows as massage to be classified under the

passive movements. Massage cannot properly be termed movement, but manipulation; and, although being intimately connected with movements, both in effect and application, it is thought best to be considered separately, as is now done by modern writers. This part is also treated very superficially, and for better information recourse must be had to special works on this subject.

The book contains a full list of gymnastic prescriptions indicated for various disorders. H. V. BARCLAY.

SYRINGO-MYELIA. By Guy Hinsdale. *Internat. Med. Magazine*, Nov. 1896.

A very able résumé of the subject of cavity-formations in the spinal cord, illustrated by reproductions of cord sections, is given by Hinsdale. The history of the disease, its method of development, its pathologic characteristics and the varieties of clinical symptoms are accurately noted. The various forms and locations of the lesions in different and in single cases are shown, as are also photographs of the arthropathies so frequently occurring in this malady. The essay is not concluded. We await with interest whatever conclusions Hinsdale may reach, for, so far, the article referred to gives us nothing original to its author, though it was the Alvarenga prize essay of the College of Physicians of Philadelphia for the year 1895.

STERNE (Indianapolis).

CLINICAL AND THERAPEUTICAL RESEARCHES ON EPILEPSY, HYSTERIA, AND IDIOCY. Report of the service at the Bicêtre for the year 1895, by Bourneville.

This work is a report upon the methods of care and teaching, the treatment and its results in the department for idiotic, epileptic and backward children at the Bicêtre for the year 1895. It opens with an account of the general arrangement of the service for the year, and the classifications of the different groups of children. All the children do such gymnastics as are suited to their age and physical ability. Those of filthy habits have careful toilet lessons. The more advanced are taught manual dexterity in various fashions, exercises in speech and in the uses of the senses, minor object-lessons and such primary instructions as can be given to the deficient.

It is interesting to see how large a part in the education, music, games, dancing and singing are made to play. A second part of the book is occupied with very full clinical descriptions of cases and pathological studies where these were made. The latter portion of the book contains reports on various methods of medical treatment. Among these is a careful one of the treatment of three cases of myxœdema with thyroid, all of which improved. It is curious and may prove valuable to know that the reaction to the thyroid in the form of the various disagreeable symptoms which accompany full doses, viz., tachycardia, feebleness of the pulse, fever, etc., were much less severe in the youngest patient, whose age was 14 years, than in the next, whose age was 20 years, and much less in him than in the oldest one, whose age was 30 years. A separate note is made of the effect of thyroid on obesity, and here again the results were favorable; and another elaborate section deals with the value of monobromate in a number of different disorders. Bourneville finds it especially useful in genito-urinary disorders and in epilepsy of the vertiginous type.

MITCHELL.

GENIUS AND DEGENERATION. By Dr. William Hirsch. 1891. D. Appleton & Co., Publishers. \$3.50.

This work is a translation from the second edition of the German work and contains in eight chapters the author's views upon the fol-

lowing general subjects: *The Limits of Insanity, The Psychology of Genius, Genius and Insanity, Degeneration, Influence of Education upon Genius, Secular Hysteria, Art and Insanity, Richard Wagner, and Psycho-pathology.*

The author's preface refers to the universality of scientific information and the large number of people interested in the subject treated in this volume, and refers to recent numerous and lengthy publications upon the allied themes of genius and insanity attesting to its great public interest.

The present volume is a worthy addition to the series. In its opening pages the work seems formal and stiff, if not academic in style; the evident desire of the author to write a popular work in which a difficult subject should be expressed in simple terms has given it a somewhat stilted character. Yet the purpose is accomplished; the illustrations are simple, and the facts and inferences are clearly and well put. The author is quite emphatic in his use of the word "genius," claiming it to be a radical error in those writers who have tried to make of it a thing "*sui generis*," and as such to be analyzed. Genius is to him a term of popular phraseology, and, therefore, not a thing upon which we may generalize too broadly.

The chapter upon the Psychology of Genius does not seem to the writer to be an up-to-date exposition of the matter, though the general conclusions are common sense ones. The analysis of poetical expression, on page 53, is rather commonplace, and in his exposition of the musical theory it appears that the cart is put before the horse, and one might imagine that he was reading the opinions of a literary critic of the flowery order instead of those of a psychologist. The author employs the opinions of early-day philosophers; but who would accept Richter, Wagner or Goethe in a scientific, psychological analysis at the present day, and yet he uses their opinions in building up his argument.

In the chapter upon Genius and Insanity the border lines are glanced at, but the author does not always use clear language, and at times even begs the question by his use of the terms, "well-proportioned development," since what the well-proportioned development may be, is the cardinal point of the problem; and when he states in the same paragraph that there is no norm what can "well-proportioned" mean, if there be no constants by which a proportion may be determined.

The chapter also contains a more or less rambling discussion of Lombroso's views regarding some of the "symptoms" found in great men and in the insane.

The inferences that psychiatrists and pseudo ones have drawn from observations, which in the main are true, should not be laid to Lombroso's door, because he first pointed out the facts. The interpretations of the facts are purely anthropological and are, moreover, complicated, and the popular treatment here given serves more to cloud than to clear the subject. Until we know more of the psychology of the emotions, popular discussions seem fruitless.

In the following chapter on Degeneration, which is excellent, the author is more at his ease. It reads not unlike a refined chapter of Nordau. Dr. Hirsch seems to make degeneration almost a synonym of insanity, yet the necessity for a stricter demarcation is pointed out.

The Influence of Education upon Genius is a particularly good chapter. A rational and optimistic stand is taken, and the matter forms a valuable contribution to the theme of the influence of education upon moral character. This chapter deals with the general question of infant prodigies and hot house education schemes in a manner which is quite in accord with the more rational thought of the everyday practitioner.

In the chapters upon Secular History and Art and Insanity, the author enters the arena to controvert the opinions of Nordau, that the present age is one of "distributed and vast hysteria." He points out that Nordau is wrong in his facts if he supposes that the popular delusions of the middle ages were less distributed or of a minor importance than some of the hysterical epidemics of to-day. He does not deny that the age is not what it should be in this matter, but he states emphatically that it is better than it was, and is still convalescing. The chapter is full of good thrusts which the erratic and extravagant opinions of Nordau deserve.

The chapter upon Art and Insanity is a masterly one. In it the question is put, How far can we from a work of art or literature diagnose a mental disease in its author? The verdict rendered is that in the world of the decorative arts, or of music, no definite conclusions can be drawn, save, of course, in some extreme cases; but with writers it is otherwise, as a deeper view into an author's psychical state may be obtained and a diagnosis made; but the reasoning should be clear and cautious before one draws inferences as to mental unsoundness.

In the last chapter on Wagner and Psychopathology the uncontrollable ecstasy of the German hero worshipper is seen. Wagner being a musician and German, Nordau, therefore, committed sacrilege in classing him among the degenerates. Our author has little difficulty in refuting the more short-sighted of Nordau's criticisms, and he sums up the chapter well.

On the whole the book is excellent. While personally the writer found Nordau's work more interesting, suggestive and amusing, the answer, or refutation, for such it really is, is more scientific, sober-minded and optimistic.

JELLIFFE.

DE L'APHASIE SENSORIELLE. [Sensory Aphasia.] By Charles Mirallié. Paris: G. Steinheil. 1896.

Aphasia, as presented by the school of Dejerine, has some peculiar features, and Mirallié, a former interne of Dejerine, has given us in this work the views of his teacher and his own observations. The book must be regarded as one of the most valuable recent contributions to this subject. The statements are clearly expressed, and no doubt exists as to the meaning of the writer. The material has been obtained from the Bicêtre, where Broca pursued his studies on aphasia, and from the Salpêtrière.

Wernicke believed that the speech centres are subordinate to one another, and there is but one form of sensory aphasia, though the word-blindness may be greater in one case and the word-deafness in another, according to the location of the lesion. Charcot, on the other hand, taught that these centres are independent, and that a lesion of any one of the four may cause a "pure" form of aphasia. Mirallié follows the teaching of Wernicke and Dejerine.

The ability to read one's name, or to understand it when heard, is not a fair test of word-seeing or word-hearing. The name makes a deeper impression than any other word. The centres for music are probably very near those for speech, but amusia may exist without aphasia. In alexia words may be recognized as designs. One of Dejerine's patients did not know the meaning of the letters R. F. until a line was drawn about them, when they became symbolical of the République Française. Another understood the secret system employed for marking goods in his store. The memory of the letters may be preserved after that of words is lost. Optic aphasia and mental blindness, according to Mirallié, have only been observed in sensory aphasia. "Jargon aphasia" is the form of speech disturbance usually seen in sensory aphasia. The patient utters twenty words where one would do, and many of these he coins. True paraphasia is very rare. In

this form every word is well known, but the combination conveys no meaning. As, usually, there is no paralysis of the limbs in sensory aphasia, the writing is easily tested, and agraphia or paraphagia is detected. The patient copies line for line, each letter is a design which he reproduces exactly. In motor aphasia the patient copies printing as script. Hemianopsia only occurs when the lesion has penetrated deeply enough to cut the optic radiation. The intelligence suffers more in sensory than in motor aphasia, as does also the pantomime, and the prognosis of recovery is not so good. In children aphasia, even sensory, usually disappears.

In the form of aphasia known as "pure" (subcortical) the zone of language, embracing the area of Broca, the left angular gyrus, and the posterior part of the left first temporal convolution, is intact, and is only deprived of one of its connections with the motor, visual or auditory cortex. Only one part of speech is affected. In "pure" word-blindness the patient may be able to read if he traces the letters with his fingers, as in this way he is able to arouse the visual images. The lesion in "pure" word-blindness is found in the calcarine fissure, lingual, and fusiform lobules, and is always associated with hemianopsia. In "pure" motor aphasia the patient has lost the power of articulation; he can understand what is said or written; he can indicate the number of syllables in a word, and can write correctly with his left hand. The transcortical motor aphasia is a form described by the Germans. The articulation of repeated words or of song is better than spontaneous utterance. It is only a stage of amelioration in cortical motor aphasia. Transcortical sensory aphasia is also theoretical.

Mirallié devotes special attention to the subject of the graphic centre. Wernicke, Kussmaul, Lichtheim, Gowers, Dejerine, Freud, Oppenheim, and others, have denied the existence of this centre. A person writes, says Mirallié, by reproducing on paper the visual images stored up in the angular gyrus, and a lesion of any part of the speech zone causes agraphia or paraphagia. Charcot believed that a word consists of four elements: the visual, the auditory, the articulatory and the graphic. A person may use one of these almost to the exclusion of the others in the processes of thought. If this theory were correct, a lesion in the speech zone would produce different symptoms, according to the predominating centre of the patient. Mirallié believes that an educated adult employs the different centres in speaking or writing in the same way as does a child, but the act has become unconscious through repetition. There has never been a case of "pure" agraphia. The case of Bar is the only one known in which the lesion was confined to the posterior part of the second left frontal convolution, and the patient had motor aphasia as well as agraphia. Any destruction of Broca's area causes agraphia. It has been demonstrated by the autopsy that a lesion of the angular gyrus produces agraphia. Mlle. Skwartzoff reports the case of a patient who would only write spontaneously the words he could read and speak spontaneously. If Charcot's theory be correct, it is necessary to believe that exactly the same images in the graphic, visual and articulatory centres had been destroyed. A person can write with almost any part of the body, the graphic centre, therefore, would have to occupy all the motor zone. If such a centre exists, there must be similar ones for piano-playing, type-writing, etc. This centre is supposed to contain graphic images, and is not merely motor; for agraphia, according to Charcot, is aphasia of the hand; is amnesia of graphic images. If there is a graphic centre, a lesion of this should affect all forms of writing, for the same movements are made in copying as in spontaneous writing. A motor aphasiac cannot write spontaneously, but can copy printing as script. This is an intellectual act, and the graphic images are used—if they exist. Why, then, are they lost for spontaneous writing? Left-handed

persons use the left cerebral hemisphere in writing and become aphasic in left hemiplegia, but the power of writing with the right hand is lost. Must we believe that the hemiplegia and motor aphasia are the result of a lesion in the right, and the agraphia of a lesion in the left hemisphere? Agraphia, says Mirallié, is due to alteration of the word-percept. A motor aphasiac cannot spell better with lettered blocks than he can write; he has, therefore, no loss of graphic images.

Mirallié records sixty-two cases of sensory aphasia; most of these have been previously published, though some have been studied by himself. An important case is given at length. SPILLER.

REVUE DES SCIENCES MEDICALES. [Review of the Medical Sciences.] January, April, July and October, 1896. Paris: Masson et Cie.

The four annual numbers of the *Revue* contain an immense number of useful abstracts and an extensive bibliography in addition. While the proportion of neurological and psychological matters treated is not very large, what is given is well done, and American contributors to the subjects seem to have received their due share of credit.

MITCHELL.

DIE SYPHILITISCHEN ERKRANKUNGEN DES GEHIRNS. [The Syphilitic Diseases of the Brain.] By Prof. Dr. H. Oppenheim, Berlin. Nothnagel's specielle Pathologie und Therapie. Vienna, 1896.

It was not at first intended to include a monograph by Oppenheim on cerebral syphilis in Nothnagel's system, but no one will regret the change of plan. Such diseases as tabes and general paresis, which are supposed to be due in some unknown way to syphilis, are not discussed in this work. A few of Oppenheim's most important statements are as follows:

Contrary to an opinion once held, it has been shown by recent investigations that syphilis of the central nervous system may develop within the first year, or even first six months after infection, and occasionally in advance of the roseola. The knowledge of this fact is due to more exact observation, and yet Oppenheim believes that the nervous system is affected more rapidly now by the syphilitic virus than in former years. It seems that the milder forms of constitutional syphilis are more apt to involve the nervous system than the severe, and, indeed, cases occur in which the only specific lesions found are in this system. Usually the signs of hereditary syphilis develop early in life, though it is not uncommon for them to be first detected at the period of puberty, or even later.

As the gumma probably grows from the vessels or connective tissue of the meninges, it is usually found either in the meninges or the peripheral layer of the brain, though it may be located within the medullary substance or central ganglia. Even in these portions the origin of the growth is in the pial processes or vascular walls. Gummata more commonly are found at the base of the brain, and especially in the interpeduncular space, and on the optic chiasm, and when they occupy the convexity, they are more frequently located on the frontal and parietal lobes. Cerebral syphilis, as a rule, is a diffuse process. Some observations have shown that primary syphilitic neuritis of one or more cranial nerves, or of many of the spinal roots, may occur. The vessels, also, may be independently affected, and the circle of Willis is especially liable to syphilitic disease. In one of Oppenheim's cases a circumscribed lesion of the basilar artery was the only manifestation of cerebral syphilis. All authors do not believe that the fatty degeneration and calcification, which are found in endarteritis deformans, are absent in the syphilitic form. Syphilis is the cause of a large proportion of the aneurysms of the cerebral arteries.

Many writers have agreed that it is difficult, or even impossible,

to make a positive diagnosis of syphilis from the histological changes in the central nervous system. Important diagnostic points are: the tendency of the process to involve the arteries of a certain calibre or only portions of these; the youthful age of the patient, and the syphilitic lesions of other organs. Certain forms of sarcomatosis may resemble syphilis, and although circumscribed tumors may be found in this process, they do not, as a rule, present caseation or the characteristic lesions in the arteries of the circle of Willis, and the process does not tend to spread from the meninges to the brain and cord. More difficult is the diagnosis between syphilis and tuberculosis. The presence of the tubercle bacillus is, of course, decisive. A purulent exudate in the meninges, or in a tumor, is almost always tuberculous, or, at least, some process not syphilitic. In a few cases in which pus has been found in syphilitic meningitis, it was probably the result of a mixed infection. Miliary tumors are usually tuberculous. Obliterating endarteritis of the basal vessels, with intact meninges, is more common in syphilis, though this condition may be found in tuberculosis. The most reliable sign is the presence of tuberculous lesions in other portions of the body, and yet patients with syphilis of the nervous system may die from tuberculosis. Syphilis may appear as meningitis without tumors, or as pachymeningitis hæmorrhagica, or as encephalitis and softening not due to vascular disease or new growths. It may cause inflammation and degeneration of the nuclei of the cranial nerve. The syphilitic virus may, undoubtedly, cause general paresis, and Moeli, even in true cerebral syphilis, has found destruction of cortical fibres. The same cerebral changes occur in the hereditary as in the acquired form of syphilis.

The nerves arising in the posterior cranial fossa are less apt to be affected, as the meningitis is not found in this part of the brain as frequently as on the pons and cerebral peduncles. Focal symptoms may be due to the growth of the syphiloma into the brain, but more commonly they are the result of vascular disease, and hemiplegia produced in this way usually develops in successive attacks. Headache is an early and almost constant symptom of syphilitic cerebral meningitis, and in the basal form it is usually felt deep within the cranial cavity. The headache is often associated with vomiting, and its severity may interfere with sleep, though cerebral syphilis may cause insomnia independently of the cephalalgia. It is not usual in basal syphilis to find the gradually increasing stupor seen in brain tumors, but the stupor occurs in attacks, though cases have been reported in which a fatal coma was the only symptom of cerebral syphilis. Drowsiness is a common symptom, and delirium also occurs. Polydipsia and polyuria are not uncommon symptoms of basal syphilis, and usually in such conditions there is involvement of the second and third nerves, and these may be the only cranial nerves affected. Oppenheim observed polyuria and polydipsia in eleven out of thirty-six patients who were believed to be suffering from basal syphilitic meningitis. It is not necessary that there should be an anatomically demonstrable lesion of the oblongata or vagus for the production of these symptoms. In two of Oppenheim's patients the meninges of the interpeduncular space were affected, but there were no evidences of syphilis in the posterior fossa. The interpeduncular space is so commonly the location of basal syphilitic meningitis that Ricord has called the paralysis of the third nerve "the signature of syphilis." Vision may be impaired when nothing abnormal is seen with the ophthalmoscope, but a permanent and complete blindness is rare in syphilis. Rapidly developing amaurosis, with a normal appearance of the fundus, may simulate hysteria. Rigidity of the pupil to light is also observed in basal syphilitic meningitis. The third nerve may be entirely penetrated by gummatous tissue, and yet only a few muscles supplied by this nerve may

be paralyzed. Ptosis from unknown cause should always suggest syphilis.

The apoplectic insult may be the first sign of cerebral syphilis. The typical course, however, is subacute with remissions and exacerbations. The arteritis of the Sylvian artery not infrequently causes thrombosis, usually with premonitory symptoms, such as headache, vomiting, vertigo, etc. The formation of the thrombus is a slow process, and there may be first weakness of one side, which later becomes paralysis; or one limb may be paralyzed, and after some hours the other on the same side may be affected. A similar mode of development of hemiplegia may occur in senile arteriosclerosis, and is not pathognomonic of syphilis, but symptoms, such as weakness, or aphasia, resulting from closure of vessels for hours or days, are very characteristic of syphilis. There have been cases of cerebral syphilis with the symptom-complex of general paresis, which has been due to a general involvement of the cerebral arteries. As a rule, disseminated sclerosis may be distinguished from cerebrospinal syphilis, but Oppenheim has occasionally found the differential diagnosis impossible. Cerebrospinal syphilis may present the symptom-complex of combined systemic disease, but the presence of cerebral symptoms, or the peculiar course of the disease, may make a diagnosis possible. There are cases of cerebral syphilis which cannot be distinguished from general paresis, and which, indeed, become cases of paresis.

Oppenheim has observed that cerebral syphilis assumes an especially serious aspect when the early manifestations have not been treated. The prognosis depends, to some extent, on the general condition of the patient, and alcoholic excesses and head injuries favor the development of the disease. The prognosis is also better as long as the process is extracerebral, *i. e.*, in the meninges and nerves, and not in the vessels. Oppenheim states that in seven persons with cerebral syphilis, treated by him, there had been no relapses for five to ten years after recovery. In his opinion it is not safe to depend on the iodide alone, but mercury should also be employed. He usually gives two to five grammes of iodide of potassium daily to an adult, and soon increases the dose, even to six or ten grammes daily. He employs three to six grammes of mercurial ointment, and stops after thirty or forty applications, though he thinks it is wise to repeat the treatment three to six months later, even if the symptoms have disappeared; and once yearly for two or three years.

SPILLER.

A Handbook of Medical Climatology, by S. Edwin Solly, M. D., M. R. C. S. Illustrated in black and colors. Lea Bros. & Co

Miscellany.

THE SECTION ON NEUROLOGY AND MEDICAL JURISPRUDENCE OF THE AMERICAN MEDICAL ASSOCIATION.

Elaborate preparations have been made in order to give didactic and clinical instruction during the week preceding and that following the semi-centennial meeting of the American Medical Association, to those members of the Association who choose to avail themselves of the opportunities offered. Clinical, laboratory and ward demonstrations will be given at the various medical schools and hospitals of the city. A special meeting of the Philadelphia Neurological Society will be held, and a number of interesting cases and papers will be presented. The programme of the Section is as follows:

Tuesday, June 1st—Chairman's Address, Dr. W. J. Hertman, Ann Arbor, Mich.; History of the Section on Neurology and Medical Jurisprudence, Dr. J. G. Kiernan, Chicago; History of American Neurology, Dr. C. H. Hughes, St. Louis; On the Pathogenesis of Locomotor Ataxia, Dr. L. Harrison Mettler, Chicago; Trunk Anæsthesia in Locomotor Ataxia, Dr. Charles W. Burr, Philadelphia; The Paralysis, by One of the Many Paralytics, Dr. Samuel Knox Crawford, Chicago; Internal Cerebral Meningitis Chronica, Dr. E. S. Pettijohn, Alma, Mich.; The Differential Diagnosis between Cerebral Syphilis and General Paresis, Dr. Hugh T. Patrick, Chicago; Hereditary Lateral Sclerosis, Dr. Augustus A. Eshner, Philadelphia; A Case of Thomsen's Disease Complicated by Multiple Neuritis, Dr. M. Nelson Voldeng, Des Moines, Iowa; Pain Traumatism, Dr. Thomas H. Manley, New York City; Melancholia and its Treatment, Dr. W. S. Watson, Fishkill-on-Hudson, N. Y.

Wednesday, June 2nd.—Aphasia, Dr. Charles K. Mills, Philadelphia; Discussion, Drs. F. X. Dercum, Hugh T. Patrick, William G. Spiller, Barney Sachs, J. J. Putnam, C. W. Burr and W. J. Hertman; French and Motor Aphasia in a Polyglot, Dr. F. Peterson, N. Y. City; The Subconscious Mind, Clark Bell, Esq., New York City; Some States of Disturbed Consciousness, Dr. J. T. Eskridge, Denver, Col.; Influence of Hypnotic Suggestion, upon Physiological Processes, Dr. R. Oscar Mason, New York City. Expertism, Dr. S. V. Clevenger, Chicago; A Synopsis of the Duestrow Case, Dr. L. Bremer, St. Louis; The Medico-Legal Aspect of Choreic Insanities, Dr. C. C. Hersman, Pittsburg, Pa.; Insanity and Pulmonary Consumption Among the Negro Population of the South Since the War, Dr. Thomas J. Mays, Philadelphia; Remarks on the Curability of Insanity, Dr. John Puntton, Kansas City, Mo.: (a) Alcohol as a Causative Factor in Disease of the Central Nervous System. (b) Inebriety and Tuberculosis as Allied Diseases, Dr. T. D. Crothers, Hartford, Conn.; The Status of the Present Treatment of Alcoholism, Dr. J. K. Bau-duy, St. Louis; Stigmata in Young American Degenerates, Dr. Eugene S. Talbot, Chicago;

Microscopic Findings in a Case of Trauma of the Cervical Region of the Spinal Cord. Dr. J. H. Lloyd, Philadelphia; Meningomyelitis with Special Reference to the Tubercular Form, Dr. William G. Spiller,

Philadelphia; Subject un-announced, Dr. Henry W. Coe, Portland, Oregon.

Thursday, June 3rd.—Neurasthenia Essentialis and Neurasthenia Symptomatica, Dr. F. X. Dercum, Philadelphia; A Study of the Symptomatology of Neurasthenia in Women, Dr. Louis F. Bishop, New York City; Clinical Evidences of Neurasthenia as an Abdominal Neurosis, Dr. G. Betton Massey, Philadelphia; Function of the Nerve Cell, Dr. Wm. B. Hall, Jr., Sewanee, Tenn.; The Causative Factors in Disease of the Central Nervous System, Dr. Geo. H. Rohe, Sykesville, Md.; The Use and Abuse of Electricity in the Treatment of the so-called Neuroses, Dr. L. Harrison Mettler, Chicago; The Rest Cure, Dr. Landon Carter Gray, New York City; Discussion: Drs. Chas. K. Mills, E. S. Pettyjohn and C. H. Hughes; Rest and Northern Lake Air for Neurotics, Dr. E. S. Pettyjohn, Alma, Mich.; Treatment of Graves' Disease, Dr. Herold N. Moyer, Chicago; Discussion: Drs. A. A. Eshner and C. H. Hughes; Habit Spasms of Children, Dr. Samuel J. Fort, Ellicott City, Md.; A Study of the Development of Some Common Psychoses of Childhood into Permanent Criminal Tendencies, Dr. J. Francis Calif, Middletown, Conn.; Hypnotism in the Treatment of Disease, Dr. U. O. B. Wingate, Milwaukee.

Friday, June 4th.—Rumination in Man, Dr. Wharton Sinkler, Philadelphia; *Tumor of the Spinal Meninges, Drs. Chas. K. Mills and Aloysius O. J. Kelly; *(a) Fibroma of the Dura, (b) Syphiloma of the Dura, (c) Glioma of the Thalamus, Drs. Chas. W. Burr and Aloysius O. J. Kelly; *Tumors of the Cerebellum with the Report of a Case, Dr. Aloysius O. J. Kelly; *A Clinical and Pathological Report of a Case of Chronic Progressive Non-specific Dementia with Arteriosclerosis, Drs. Chas. K. Mills and Mary Alice Schively; *A Case of Paralytic Dementia with Autopsy, Drs. Chas. W. Burr and J. H. W. Rhein; Tumor of the Basal Ganglia, Drs. Charles W. Burr and Carl Ohnberg; *Tumor of the Spinal Meninges, Drs. Samuel W. Morton and A. Ferree Witmer; Bilateral Psychomotor Myo-Synchrony, Dr. C. H. Hughes, St. Louis; (a) A Contribution to the Pathology of Myelitis, Acute and Chronic, (b) Lesions of the Spinal Cord Due to Tubercular Disease of Column, With Microscopic Specimens, Drs. John K. Mitchell and John H. W. Rhein, Philadelphia; Tremor in Chorea, Dr. John H. W. Rhein, Philadelphia.

From the Second Report of the Neurological Laboratory of the Philadelphia Polytechnic.

Members of the Section are invited to visit the Laboratory.

INTERNATIONAL CONGRESS OF NEUROLOGY, PSYCHIATRY, MEDICAL ELECTRICITY AND HYPNOLOGY.

(Corrected Announcement.)

First Session—Brussels, from September 14 to 19, 1897. International exhibition of Brussels.

Honorary President, Mr. Schollaert, Minister of Interior and Public Instruction; President, Professor Verriest, of Louvain; Vice-Presidents, Professor Van Gehuchten, of Louvain, and Dr. Lentz, Director of the Asylum for Insane of Tournay; General Secretary, Dr. Crocq (son), Substitute of the Faculty of Medicine M.D. at the Hospital of Molenbeek; Secretaries of the Sitzings—Neurology, Doctors Glorieux and Mahaim; Psychiatry, Doctors Claus and DeBuck; Medical Elec-

tricity, Doctors Libotte and Swolfs; Hypnology, Doctors L. Democ and Van Velsen.

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Questions Submitted for Discussion—(a) Neurology: 1. The surgical treatment of epilepsy—Its indications and consequences; Reporter, Prof. Winkler, of Amsterdam (Holland). 2. Pathogeny and treatment of exophthalmic goitre; Reporter, Prof. Eulenburg, of Berlin (Germany). 3. Pathogeny and semeiology of reflexes; Reporter, Mendelssohn, of St. Petersburg (Russia). 4. (Subject to be selected) Reporter, Prof. Oppenheim, of Berlin (Germany). 5. Influence of the delivery on the nervous and mental diseases presented later by children; Reporter, Prof. Anton, of Graz (Austria-Hungary). 6. Pathogeny of the muscular rigidity and of contracture in the organic affections of the nervous system; Reporter, Prof. Van Gehuchten, of Louvain (Belgium). (b) Psychiatry: 1. On the diagnostic value of the prodromic symptoms which long precede the manifestations of general paralysis; Reporter, Prof. Thomson, of Bonn (Germany). 2. Psychoses and dreams; Reporter, Dr. Sante de Sanctis (Roma). 3. On the modifications of the morbid type of progressive paralysis of insanity during the thirty last years; Reporter, Prof. Mendel, of Berlin (Germany). 4. On the relation between the psychoses, mental degeneration and neurastheny; Reporter, Dr. Lentz, of Tournai (Belgium). (c) Medical Electricity: 1. The semeiologic value of electrical reaction of muscles and nerves; Reporter, Prof. Doumer, of Lille (France). 2. The therapeutical value of high frequency currents; Reporter, Prof. Bergonié, of Bordeaux (France). (d) Hypnology: 1. The therapeutical value of hypnotism and suggestion; Reporter, Dr. Milne-Bramwell of London (England). 2. The question of criminal suggestions—Their origins and actual state; Reporter, Prof. Liegeois, of Nancy (France).

GENERAL SECRETARY,

27 Palmerston Avenue, Brussels.

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Original Articles.

AN UNUSUAL CASE OF HEMIPLEGIA.

By WM. G. SPILLER, M. D.,

(From the Clinic of Dr. S. Weir Mitchell).

C. P., aged thirty-nine years, is engaged at the Presbyterian Hospital. There were no organic nervous diseases and no neuroses in the families of the parents of the patient, and his parents did not have rheumatism.

The patient was born in Denmark, and has been in America about twenty-five years. Before his attack he could speak fluently Danish, Swedish, Norwegian, German, French, Dutch and English, and could understand a little Italian and Spanish. He was chief proof reader on the Philadelphia Press.

On the 14th of August, 1891, after bathing in the ocean at Atlantic City, he noticed two men in the water beyond their depth, and in danger of drowning. He plunged into the water; reached the men, and supported one with his left arm, while the other clung about his neck. He was in this position about twenty minutes before the life boat came and the men were picked up. The patient was left to rescue himself as best he could, for he was not supposed to be in danger. He was seized by the undertow; was upset and so exhausted that he was unable to swim, and was obliged to float to the shore. The same afternoon he took the train to Philadelphia, and *en route* wrote a letter to his wife in Denmark telling her of his vacation. After

returning to Philadelphia he worked the whole night reading proof. On the morning of the 15th of August, about six o'clock, he went home; told his landlady about the events which had occurred at Atlantic City; went up-stairs to go to bed and undressed, but before he got into bed he lost consciousness and fell to the floor. He was unconscious a very few minutes, and is sure of the correctness of this statement, as he noticed by the clock that only about five minutes had passed. When he regained consciousness he found he was unable to rise, though he could raise both arms and move his head from side to side. No movement in the lower limbs was attempted. While raising and lowering his arms he found that the power of motion became gradually less, and that he raised his arms to a lower level at every attempted movement, and finally that he could not move them at all. After he had lost all power in both arms he said aloud in English: "Now, here's a pretty go." He remembers this perfectly, as they were the last words he uttered for some time. He laid meditating on his condition and believes he may have become unconscious again, or simply have fallen asleep. He regained consciousness about eight or nine o'clock on the same morning, and tried to give an alarm, but could not make a sound (aphonia), and could not move any part of either arm, either leg, or of the fingers. He does not know whether he had any power of motion in the muscles of the head. He fell asleep and awoke from time to time. About three o'clock he heard people talking at his door and could understand every word, but could not say anything. Some persons entered his room, lifted and placed him in bed. He was asked to which hospital he wished to be taken, and is said to have replied: "German Hospital," though he had never heard of this place. He remembered the events which occurred *en route* to the hospital, but lost consciousness after entering the building until Monday afternoon, August 17th, 1891, about five o'clock. When he regained consciousness about this time, he found he could use his left limbs, but could not move his right arm and leg, nor fingers of the right hand; and he is quite positive that the right side of the face was paralyzed. There was anesthesia over the right side of the face, trunk, and in the right extremities, and over the right side only of the genitalia. This anesthesia was sharply defined by the me-

dian line. A pin stuck into the flesh a quarter of an inch was not felt. Sensation was lost for about a month, and recovered gradually.

After the patient awoke on the Monday following the onset of the paralysis, he had numerous fantastic dreams in which water played a chief rôle. He saw men and women dancing on the water, and he himself took part in this performance. He thought he swam to different islands and saw new faces everywhere. These statements show the deep mental impression which his rescue of the two strangers had produced.

On Tuesday or Wednesday of the same week his friends came to see him; spoke to him in English, and he replied in Danish, his native tongue. Aphonia, therefore, lasted only a few days. He had left Denmark when a boy of fourteen or fifteen, but had made frequent visits to that country. He could understand every word said in English or German and some in French, but could only reply in Danish. He remained in the hospital three months, and before he left he could say some sentences in English or German as well as in Danish, but could not speak fluently. These were the only languages he had heard since the beginning of his attack, with the exception of a little Swedish. A Swedish minister called to see him and spoke to him in the Swedish language. At first the patient could not understand a word said to him, but suddenly he found he could comprehend though he could not reply in Swedish. In 1894 he went back to Denmark and visited Sweden for a day and a half, and during this short visit he regained the power of speaking the language of that country, though he does not remember having spoken it since his misfortune began. He has not had occasion to speak Dutch and French since he was paralyzed, and cannot well recall these languages, but probably would be able to do so if he heard them spoken. He does not think he tried to write until a year and a half after the commencement of the paralysis, and then wrote a letter in English with his left hand. Every word was said by his fellow proof readers to be correctly spelled and punctuation was perfect. He could also write in Danish, but in no other language. He is able to understand the meaning of the French he reads, though he does not recognize the individual words. When a sentence was spoken in this language he said: "It sounds familiar, but I do not know what it means."

The patient now presents the appearance of a vigorous, intelligent man. He makes no use of his right arm. The circumference of the right arm measures 12 inches; of the left, $12\frac{1}{2}$ inches; of the upper part of both forearms, 11 inches; of the right hand, 8 inches; of the left $8\frac{1}{4}$ inches. There is a slight tendency to flexor contracture of the fingers of the right hand. The skin of this hand is thinner and the hand is somewhat flabby. The fingers of this hand are more flexible on rising in the morning. He has no power of extension, abduction or adduction in the fingers of the right hand, and only slight power of flexion. On raising the right arm, which he can only do to a very limited degree, a violent intention tremor is developed in the arm, forearm and hand. Forcible extension of the hand at the wrist also causes this tremor, but an attempt to passively flex the forearm on the arm tends to partially check it. The tremor at the wrist partakes at times of the movements of flexion and extension; at other times it is more rotatory in character. There is no tremor when the limb is at rest. The patient has had this tremor in the arm for two or three years; he had a similar tremor in the right leg, (though this was observed also during rest), which developed soon after the paralysis and ceased gradually three years ago, though it returns even now when he is excited. This involuntary movement has not been observed in any other part of the body. Sensation for heat, cold, pain and touch is now about the same on the two sides of the body. On rising in the morning he can walk almost without a limp and can speak more fluently. In walking he swings the right leg. There is no muscular atrophy in any part of his body. The knee jerk and muscle jerk in the right lower limb are exaggerated, and ankle clonus is present on the right side only. The reflexes at the wrist, olecranon and biceps tendon are exaggerated on the right side. The reflexes on the left side of the body are about normal. The chin jerk is prompt. There are no hysterogenic zones. The nasolabial fold on the right side is a little less prominent than on the left. The tongue is protruded in the median line. There is no limitation of the fields of vision, as tested with the fingers. Taste, smell and hearing are normal. There is no history of venereal disease to be obtained, though his wife has had two or three miscarriages. The patient has three living and healthy children.

It was thought for many years, even by Charcot, that paralysis of the muscles innervated by the seventh nerve does not occur in hysteria. That this is a mistake the following quotation from Charcot's works shows: "He presents an example of evident and real hysterical facial paralysis the existence of which, until recently, has seemed to me very doubtful. The paralysis . . . is slight, without doubt, but is very evident" (*Clinique des Maladies du Système Nerveux*. Notes collected by Georges Guinon, 1892, p. 290. See also the *Traité Clinique et Thérapeutique de l'Hystérie*. By Gilles de la Tourette. Second Part).

The presence of ankle clonus may be considered by some as opposed to the diagnosis of hysteria. Sternberg, in his elaborate monograph on the tendon reflexes, says: "In hysterical persons frequently general increase of the tendon reflexes occurs, and in about 20 per cent. foot clonus is present." (*Die Sehnenreflexe und ihre Bedeutung für die Pathologie des Nervensystems*. By M. Sternberg. P. 254.)

The case, undoubtedly, at present presents the appearance of one of organic hemiplegia, but it is only possible to make a diagnosis from the history as given by the patient, and from this, it seems to me, the hemiplegia must be considered as probably hysterical.

I am indebted to Dr. S. Weir Mitchell for the privilege of studying and reporting this case from his clinic at the Orthopedic Hospital.

A CASE OF SYRINGOMYELIA FOLLOWING TRAUMA. Deutsche medicinische Wochenschrift, No. 8, 1897. By L. Huismans.

The patient fell twice on the same day (March, 1895) and struck his right elbow and shoulder. He complained constantly of pain in these parts, and in April, 1896, the right shoulder was observed to be very large. The diagnosis of syringomyelia was made in August, 1896, on account of muscular atrophy of the humeroscapular type with pseudohypertrophy on the right side, kyphoscoliosis, marked alteration of pain and temperature sense with preservation of tactile sense, paresis of the detrusor urinæ, left unilateral atrophy of the tongue, nystagmus, absence of the right patellar reflex, arthropathies, and atrophy of the small muscles of the hand. The arthropathy of the shoulder is the most common form of joint affection in syringomyelia. The writer thinks an ascending neuritis with secondary involvement of the cord, causing cavity formation, developed as a result of the two falls. The process must have extended to the oblongata and the lumbar region.

SPILLER.

THREE CASES OF THE FAMILY TYPE OF CEREBRAL DIPLEGIA.

By F. X. DERCUM.

The following cases, because of their rarity and because of the family history, are exceedingly interesting and worthy of record. They are all children. The father presents a family history which is negative as regards nervous and mental affections. The mother of the children is his third wife. He had also had a number of children by both his first and second wives; all of these children were normal. The third wife has borne him four children, three of whom present marked spastic diplegia. The family history of the third wife, the mother of the diplegic children, is interesting from the fact that the child of one of her paternal aunts is paralyzed—she thinks in a manner similar to that of her own children. She herself is perfectly healthy. Every one of her four labors was normal. No delay or incident worthy of mention occurred. The history of the children is briefly as follows:

Oscar, eleven years of age, presents the symptoms of a typical spastic diplegia. He was normal at birth and continued well up to sixteen months of age. He was able to walk unassisted, his gait seemed perfectly normal, and he was able to say quite a number of words and understand much that was said to him. At this time he was seized with a convulsion which was general, and during which he was unconscious. After the convulsions he seemed stupid, had lost largely the power of speech, and was weak and helpless. Some time afterward it was noticed that his legs were stiff, and when he began to use them, it was seen that he moved them very awkwardly. A similar condition of affairs was present in the arms. The spastic symptoms seem to have made their appearance gradually, and finally became pronounced.

At present he is able to walk, but can do so unsupported for a distance of a few feet only. The gait is spastic and tottering. The legs are quite rigid in extension. The arms are also spastic, but to a much less degree than the

legs. Athetoid movements of the hands are now and then observed. When seated the feet assume the position of talipes equino-varus. His speech is so defective that he can speak only two or three words, and these are uttered so indistinctly that they can be comprehended by his parents only. Subsequent to the convulsion he had attacks of *petit mal* during which his head would suddenly drop. At times he would fall forward. His head is much bruised from falls received during these attacks. He is exceedingly stupid and quite idiotic; he is also incontinent.

Justus, six years, was well until four years of age. Up to this period he had been a bright and apparently normally developed child. At this time he was attacked by measles, the attack appearing, however, to be one of moderate severity. During the convalescence a profound change was noted in him. He was stupid, and appeared to be weak in both arms and legs, the weakness being most profound upon the right side.

Justus presents an excessively marked spastic diplegia. Both legs are exceedingly rigid, the legs being slightly flexed at the knees, the feet completely extended. The right arm is held firmly in a semi-flexed position. The left arm is also spastic, but the patient is able to move it to some extent. He frequently attempts to walk, but invariably falls to the ground; and while in this position he writhes with choreiform and athetoid movements. At other times, when sitting in his father's lap, spasmodic movements are observed in legs, arms and face. He is also epileptic, his seizures having the character of *petit mal*. His intelligence appears to be somewhat better than that of Oscar; he talks a little more, and the words are uttered more distinctly, although bradylalia is pronounced. He is not entirely continent, sometimes not telling his mother when his bladder or bowels are to be emptied.

Cleveland, two years old, was a normal child at birth and appeared to develop in a perfectly normal manner until he was two years of age. He was then attacked by measles at the same time as his brother Justus and, like Justus, during convalescence became diplegic and epileptic. During one of his early epileptic attacks he appeared to lose his vision, but this subsequently returned. At present he has marked diplegia, but is able to walk without falling. He can, although with difficulty, pick up small objects

from the floor. Athetoid movements are present in the fingers as in the other cases. He appears also to be somewhat more intelligent than his brothers.

All of the children present tumid, sodden features like those of confirmed epileptics. Oscar and Justus look stupid and idiotic, though this cannot be said of Cleveland. In all of the cases the reflexes are exaggerated wherever the contractures and rigidity do not interfere with their being studied. In none of the cases was there nystagmus. Owing to the restlessness and poor mental condition of the children it is impossible to make a satisfactory examination of the eye ground. Vision appears, however, in all three to be somewhat defective. All of the cases drool more or less, especially is this true of Cleveland.

An examination of the remaining child, the second in age, a boy of nine and a half years, proved negative. He is in average physical and mental health, and his intelligence is fully up to the average, if not above. It was interesting to note that this child was not attacked by measles, nor has he had any other serious illness.

In these cases there seems to be present a feebleness of development on the part of the neurons of the motor area. It would seem that if not interfered with, they had the power of developing in a normal manner but that their power of resistance was so low that they underwent degenerative changes from very slight causes. In two of the cases the cause seems to have been the toxicity of measles. Freud regards these cases of family diplegia as analogues of Friedreich's ataxia, and in a sense this is true.

Cases of diplegia of the family type are very rare. A number of cases, however, are on record, among which are the following:

Pelizeaus (*Archiv f. Psychiatrie*, Vol. XVI., 1895) describes five cases of the affection occurring in three generations. All of these cases were males and presented in addition to spastic diplegia, imbecility. Gee (*St. Bartholomew's Hospital Reports*, 1889, XXV., p. 81) describes three cases. His cases are remarkable from the fact that they instance transmission of the disease through the father. The first case was thirty-seven years of age, and the father of the other two. His symptoms were those of spastic diplegia. His family history presented the follow-

ing features: a maternal aunt completely paralyzed, two maternal cousins who were born deaf and dumb, and one cousin who was born without fingers. His daughter, aged twelve, and his son, aged eleven, constitute the second and third cases described by Gee, and presented typical spastic diplegia. Sachs (*Jour. of Nervous and Mental Disease*, 1887 and 1882) reports two instances of diplegia occurring in families. First, two sisters, one of them becoming diplegic at the age of three months, and the other at the age of eight months; the former presenting spastic palsy and the latter flaccid palsy. In the second instance there were four children who after a period of normal development became spastic and idiotic. Massolongo (cited by Freud) records three sisters, who each at the age of seven years developed spastic paralysis and athetosis. Freud (*Neurolog. Zentralbl.*, 1893, XII., pp. 512 and 542) places on record the following interesting cases. A physician had married his niece, the child of his sister. Six children were born to this couple. The first was born prematurely and died soon after birth. The second and third presented spastic diplegia. The fourth child was normal. The fifth died paralyzed and idiotic. (This child Freud did not himself see.) The sixth child was born prematurely and also died early. The three children which survived were all boys. One of them was normal whilst the other two had spastic diplegia. In addition, the latter presented atrophy of the optic nerves, nystagmus and bradylalia.

DEATH IN HYSTERIA.

M. R. Le Fournier (*Thèse de Paris*, 1896) writes upon the possibility of death in hysterical troubles, and maintains that hysteria should not be regarded as a disease having always a favorable prognosis. Death sometimes takes place from violent convulsive attacks with spasm of the glottis. Anorexia occasionally causes death by inanition, and the author lays some stress on the danger of hypnotization without proper precautions, and points out that it has sometimes been known to bring on fatal spasmodic attacks.

MITCHELL.

A CASE OF PRIMARY NEUROTIC ATROPHY.

BY F. X. DERCUM, M. D., AND ISAAC LEOPOLD, M. D.

Primary neurotic atrophy is still called a rare affection, and the case before us is interesting, not only because of the typical character of the symptoms presented, but also because of the great improvement following surgical treatment, rest, massage and electricity, together with the administration of desiccated thymus gland.

S. M., male, aged 15 years. The family history is negative, save that the father suffered at one time from a profound nervous prostration. The patient himself presented no special symptoms up to eight years of age, when it was noticed that he began to drag his right foot. When ten years of age he severely sprained this foot. Subsequently and very gradually a talipes equino-varus developed, and at a somewhat later period a talipes equino-varus also developed in the left foot. The boy also noted that he could not hold things as well as other boys, that objects would tend to slip or fall from his hands. His condition, when seen by us, was as follows:

Marked talipes equino-varus of both feet, most pronounced in the right. Marked atrophy of the muscles of the right leg, especially of the peroneal group. A similar wasting, though less marked, was also noted in the left leg. The muscles of the right thigh also presented atrophy, especially the vastus internus. In the left thigh wasting was present though not decided. The patient walked with an awkward waddling gait. No atrophic changes were noted in the muscles of the trunk. The muscles of the shoulders and of the arms were fairly well developed. Those of the forearms were decidedly atrophic. This was most evident in the extensor group, and more in the right arm than in the left. It was noted that the patient allowed his hands to dangle from the wrists, the position suggesting wrist drop, although the patient was able, when requested, to fully extend the wrists. The muscles of the hands, especially of the right, were also atrophic, there

being decided wasting of the thenar eminences, flattening of the hypothenar eminences, atrophy of the interossei, and marked hollowing of the palm due, apparently, to wasting of the lumbricales. There was diminished power of extension in the hands. The grip was very weak.

Because of the great deformity, the patient was subjected to tenotomy in order to correct, as far as possible, the club foot. Before operation the case showed both



feet in the position of talipes equino-varus, with extreme shortening of the tendons of all the muscles on the posterior and external surfaces of the leg without any apparent increase in the diameter of the bellies. The plantar arch (osseous) was much increased in depth by shortening and atrophy of the muscles in this region combined with contraction of the plantar fascia. The toes were extended at the metatarsal joints, but rigidly flexed at the middle and distal joints; the intermetatarsal spaces were sunken, indicating that the interossei and lumbricales were atrophied, whereas the extensors and the flexors of the toes

were shortened sufficiently to overcome their action. The scaphoid in the right foot was displaced anteriorly and externally by the extreme shortening of the arch, and was so far removed in its articulation above and below as to make the intertarsal articulation practically useless. The most striking objective phenomenon was the peculiar waddling gait. There existed at the height of each plantar arch a point of exquisite tenderness, pressure upon which



gave rise to pain, and at the same time caused a tonic spasm of the muscles of the leg which ceased when the pressure was discontinued.

On Nov. 14, 1896, he was operated on by Dr. DeForest Willard and Dr. Leopold. The plantar fascia was first completely divided, the tendo Achillis next and, lastly, all the tendons of the toes. The adhesions being thoroughly broken, the feet were placed in a position slightly overcorrecting the deformity, and then encased in the usual immobile plaster dressing, in which they remained for three weeks.

When the dressing was removed, the feet were found in comparatively a normal position, though the toes still showed a tendency to return to the old deformity, while the arch was still somewhat high. The displaced scaphoid was, however, much less prominent, having been so far reduced as to make its articulation serviceable. There was great separation of the divided tendons—two inches in the tendo Achillis. The spots of tenderness before noted over the arch had disappeared. The gait was greatly improved, the patient using his heels with a good attempt at a forward roll of the foot. Some of the old "waddle," however, still remained. Additional treatment consisted in the administration of iron, arsenic and strychnia, also extract of thymus gland (the latter suggested by Dr. Der-cum). Daily massage, faradism, with regular exercises of various kinds under a competent instructor at a gymnasium have aided greatly in bringing about the present great improvement in the patient's condition. Shoes to fit the feet properly were made after the model taken from a cast made by Dr. Merrill Galloway, to whom we are indebted, both for casts and photographs.

Electrical examination, Jan. 14, 1897, revealed marked quantitative diminution in the muscles of the legs, but no reaction of degeneration.

Examination of the eyes revealed no symptoms worthy of record.

LATAH. A MENTAL MALADY OF THE MALAYS. W. G. Ellis. *Journal of Mental Science*, January, 1897, p. 32.

Under the term Latah, the Malays understand a peculiar nervous condition which is somewhat transitory in type and which varies so widely that rarely are two people affected in just the same manner.

Tuke's definition of the disease as a form of religious hysteria is far from the truth in the author's view. The author describes two types: (1) In these the disorder is marked by an involuntary and generally unwilling mimicry, suggestive of forms of hypnotism, but as consciousness is not affected, this is ruled out. (2) In the second type paroxysmal, short and abrupt attacks of coprolalia, are the distinguishing features.

A few cases are given in extenso, illustrating the two types.

Clinical Cases.

MARANTIC THROMBOSIS OF INTRA-CRANIAL VEINS COMPLICATING TYPHOID FEVER.

By A. B. RICHARDSON, M.D.

G. B., aged 43, married, American, white, motor man on street car, was seen in consultation with Drs. Wissinger and C. S. and W. D. Hamilton on the evening of September 9th, 1896. The following history was elicited: The patient had always been a vigorous, healthy man without evidence of specific disease, temperate and accustomed to hard work. About twelve years previously he had been injured in a railroad wreck in which the right side of the head was bruised, and there appeared to the wife to be some depression which afterwards disappeared. As far as known he had no further symptoms as a result of this injury, and was engaged in hard work for twelve years afterwards. In June last, while at work on a street car and in a stooping position, he was struck on the left side of the head by the handle of a brake being unexpectedly released. The severity of this injury is not well attested, but he was off duty for a time as a result of it, and his wife says he complained more or less of headache from that time until his last illness. On August 25th, Dr. Wissinger was called to see him and found the characteristic symptoms of typhoid fever. He had been sick about one week. He had the characteristic tongue, tenderness of the bowels, tympanites, diarrhoea, and rose spots on the abdomen. During this week, which was probably the second week of the fever, the temperature ranged from 101° F. in the morning to 103° F. in the evening. There was considerable pain in the head, but no delirium. From Sept. 2nd to Sept 7th the temperature began to decline and was about normal in the morning. On Saturday and Sunday, Sept. 5th and 6th, the pain in the head became much intensified and was concentrated in the left temporal region. On Monday the doctor was called and found him in a condition of col-

lapse. The temperature was subnormal, and he still complained of the severe pain in the left side of the head. This condition persisted through most of the day and gradually gave way under stimulating treatment, but the pain continued. The temperature on Tuesday was a degree or so above normal, and the condition of collapse had disappeared, but the head symptoms were worse. The pain was severe, the pupils were contracted, there were some somnolence and delirium, and in the latter part of the day convulsive movements were noticed in the left arm and leg and in the right side of the face. On Wednesday morning the symptoms were still more serious. There was marked coma, the pupils were much contracted, and the right more so than the left. Convulsive movements of the left side of the body were frequent, more marked in the leg. During the day convulsive movements were noticed on the right side of the body, also, and on the right side of the face.

Dr. C. S. Hamilton had been called to consider the possibility of operative interference, and I was also asked to see the case, and did so about 5 o'clock that evening in company with the physicians above named. The patient was lying on his back in profound coma with a congested, oily state of the integument, profuse perspiration, pin-hole pupils, the right more contracted than the left, and injected sclerotics. There was no response to light, the conjunctiva was insensitive, and the temperature was 103° F. There were at intervals convulsive movements in the left leg, extending to the left arm, and occasionally affecting also the right side of the face. There had been no nose-bleeding and there was no evidence of local congestion or blood stasis. After conference it was decided that the symptoms were not sufficient to justify operative interference. They were too diffuse and confusing to justify the diagnosis of cerebral abscess, and I was of the opinion that the case was one of acute meningitis. It was suggested that his eyes be examined to determine the condition of the circulation in the fundus, and during the night Dr. W. K. Rogers saw him and made the examination, which revealed a certain amount of general engorgement of the vessels, more pronounced in the left eye. He was seen again by Dr. Wissinger and myself on the morning of the 10th. His temperature was then slightly above

102° F., the pupils were still much contracted and unequal, but there was more response to light and the conjunctiva was more sensitive. He was still in marked coma and could not be aroused to respond to questions. Convulsive movements had been noticed in the left leg and arm and in the right side of the face during the night at intervals and still continued. An unfavorable prognosis was given and operation was not deemed justifiable. He died on the 12th, without any return of consciousness and with no particular change in the symptoms.

The autopsy was made on the 13th in the presence of Drs. Wissinger, W. D. Hamilton and myself. The brain alone was examined out of deference to the wishes of the relatives. On removal of the calvarium and the dura no signs of meningitis were found, considerably to my surprise. The surface of the arachnoid was smooth, there was neither pus nor lymph exudate, and no considerable increase of serum. The principal veins on the surface of the pia were distended with hard clots. One clot in particular of large size lay along the fissure of Rolando on the right side. The sinuses, however, both the longitudinal and lateral, were patulous and contained only a moderate amount of fluid blood. In the first left temporal convolution there was an area about the size of a small hickory nut within the cortex that broke open as the brain was removed and discharged a small amount of pus and some broken down brain tissue and debris of blood coagula. On the right side of the brain, just anterior to the Rolandic fissure, at about its middle and upper thirds, there was another area, not so much softened as the first, but dotted with enlarged vessels, filled with blood clots and beginning to break down. This area also reached to the cortex, but did not show externally, and extended some distance into the white substance. It was as large as a good-sized hickory nut and not well defined in outline. In the right occipital lobe there was a third area almost as large, more deeply seated, not at all softened, but dotted with vessels much enlarged and filled with hard blood clots. These were from a sixteenth to an eighth of an inch across their cut surface and very numerous. The area was not well defined. The veins of the surface of the pia were practically all filled and much distended with well formed dark clots. No signs of inflammation of the lining

membrane of the veins could be noticed with the naked eye, but microscopical examination was not made. The skull did not show any sign of injury from either of the accidents that have been recorded. The diagnosis was thrombosis of the cranial veins, probably affecting first the left temporal area and, later, the right Rolandic and occipital regions, and due to the blood stasis and blood changes in the condition of collapse during the later period of the typhoid fever. I have thought it of interest to place the case on record as in some respects it seems to be unusual and instructive.

Thrombosis of the principal cranial sinuses is not infrequent, and two well-defined varieties have been described. One, the marantic form, which occurs in exhausting diseases and states of blood dyscrasia, and is probably due to blood changes and retarded blood current; and the other infective, and due to infective inflammation of the lining membranes of the sinuses from an adjacent or metastatic morbid process. I fail to find in any authoritative work, however, much account of this condition of thrombosis occurring in the cranial veins and not in the sinuses. Gowers asks the question, "Does primary thrombosis ever occur in the veins and not in a sinus?" and answers that "there is some pathological evidence that it does." Brill, in Dercum's Text Book, does not mention it. Hirst says it "may occur in the veins as well as in the sinuses," and "the venous thrombosis may extend into the sinus and be taken for a sinus thrombosis." Ross does not mention the occurrence in the veins. Neither do Rosenthal, Gray nor Dana. Maceven goes very extensively into the subject of thrombosis of the intra-cranial sinuses in his volume on diseases of the brain and spinal cord, but says nothing whatever of the marasmic form affecting the veins alone. He says the marasmic form is much less frequent than the infective, and "is almost invariably located in the longitudinal, rarely in the lateral, and still more rarely in the cavernous sinus." All the authorities unite in the statement that it is usually found at the extremes of life in weakly persons and in the course of exhausting diseases, yet in this case, if we are correct in the diagnosis, it occurred in a strong, healthy man 43 years of age, and in the third or fourth week of a typhoid fever that had been uncomplicated, and in which the fever did not rise as high as 103° F. It would be interesting to know whether the

collapse was the cause or the result of the thrombosis. I have already said that I believed it was a contributing cause, and may it not be explained in part by the peculiar blood state that favored the thrombosis? All authorities also speak of the difficulty of diagnosis and the similarity that exists in the symptoms of thrombosis and those of meningitis and cerebral abscess. The absence of evidences of local blood stasis, they say, makes this diagnosis impossible, as the other symptoms are practically those of meningitis. In this case the absence of thrombosis of the principal sinuses caused an absence of any evidence of local stasis of the blood current, such as nose bleeding or local congestion of the integument, and thus rendered the diagnosis practically impossible. I am much indebted to Dr. Wissinger for many of the clinical data on which the foregoing is based.

TWO CASES OF TUMOR OF THE VERTEBRAL CANAL PRESSING UPON THE SPINAL CORD. By Prof. Raymond and Dr. Nageotte. *Journal de Neurologie et d'Hypnologie*, Nos. 1 and 2.

What makes one of these cases interesting from a clinical point of view is, that while there was at first spastic paraplegia accompanied by anæsthesia, the paralysis afterwards became flaccid, with loss of the reflexes, although the tumor was situated in the middle dorsal region and histological post mortem examination showed the complete absence of lesions in the lumbar portion which could have explained the loss of the reflexes.

In both cases the histological examination revealed interesting and practically important facts. At the level of the tumor where the cord was exposed to the pressure nearly all fibres on the transverse section were found degenerated. This degeneration was stated to be only in the medullary sheaths, however, while axis cylinders seemed to be intact. The authors discuss the question, whether the longitudinal extent of such lesion of the nerve fibres may be of influence in this sense, that when the lesion of the medullary sheaths reaches a certain longitudinal extent the function of the fibre becomes abolished. In the second case the said changes of the nerve fibres had affected these in a lesser height than in the first case and the sensory disturbances were more marked in the latter case. In case 1, the clinical cause would further speak in favor of the theory according to which complete transverse lesion of the cord leads not to exaggeration, but to abolition of the reflexes in parts below the lesion; if it is assumed that at first the degeneration of the medullary sheaths had not reached such longitudinal extent as to abolish their function, but that it did so in the further course.

Practically the two cases demonstrate the important fact, that after compression of the cord has lasted for some time, leading to complete sensory and motor paralysis, recovery may still be expected, if the cause is removed; since at this stage the axis cylinders of the compressed fibres may still be normal, although their medullary sheaths have become degenerated.

A CASE OF TABES DORSALIS, WITH DELUSIONAL INSANITY.

By FRANCIS O. SIMPSON, L.R.C.P., M.R.C.S.,

Pathologist and Assistant Medical Officer, West Riding Asylum, Wakefield, Eng.

The patient was a widow, aged forty-one, her education was medium and she belonged to the Salvation Army. The medical certificate upon admission stated that she had been much excited by her religious observances prior to admission and had been employed in writing articles for several periodicals. Eventually she broke up her home and gave all her household possessions away, saying she had no further use for them as the Lord would provide for her. She walked about the house with a lighted lamp in her hand, saying she was one of the ten wise virgins; the lamp went out and she then stated she had become one of the foolish ones. She is said to have had several previous attacks of "paralysis," from each of which she claims to have recovered. The patient's husband died in this institution three months prior to her admission from general paralysis of the insane. He was of intemperate habits and had a clear history of syphilis, which he communicated to the patient. The first child resulting from their union was healthy, then followed three abortions, whilst the fifth child is idiotic and shows clear signs of hereditary syphilis. The patient and her husband suffered from secondary syphilitic manifestations at the same time.

Upon his side there was a family history of insanity and intemperance; apoplexy and consumption on her's. The eldest child is described by the patient as being passionate and unmanageable, evidently markedly neurotic. Upon admission the patient had all the classical symptoms of locomotor ataxia extremely well marked, together with numerous delusions and hallucinations. She believed that she heard raps upon the table in the early morning, and these were the "power of evil" addressing her because she was not doing God's will. Stated that she was carried to a meeting in a chair before admission, that some one laid his hands upon her and she was cured by faith, being

able to walk home and subsequently to perform her ordinary household duties for a considerable time. She was quite unable to walk around the room without holding on to the table and steadying herself by grasping the other articles of furniture, but insisted upon being allowed to make the attempt, saying that the Lord would not allow her to fall.

There was paralytic mydriasis of left pupil and the right was also considerably dilated and irregular in outline. There was no reaction to cutaneous stimulation on right side, but the left responded sluggishly. Both pupils reacted well to accommodation. The optic discs were normal to ophthalmoscopic examination. There was very slight ptosis of the left upper eyelid. Sensation in the legs was much diminished and there was marked disturbance of equilibration, with much ataxia in walking, Romberg's sign being well defined and the tabetic gait very pronounced. The patient could only stand steadily with the eyes wide open and the feet placed far apart. The arms were not involved in these troubles, nor was there any loss of sensation in the upper extremities, but she suffered much from "lightning-pains" in them. The tendon reflexes were abolished in the lower but present in the upper extremities. Her mental equilibrium was exceedingly unstable and she varied between states of profound depression, with refusal of food, and considerable exaltation. A week after admission she retained the same ideas and said that she had been sent here for us to see the "finishing of the miracle" of her cure. At times she refused both food and medicine, stating that she had been ordered to do so by the Almighty as a punishment for her sins. She was incoherent and excitable. Her medicine consisted of ten-grain doses of the iodide of potash administered thrice daily.

A month later she became much troubled with girdle pains and the "lightning-pains" also became almost unbearable. The iodide of potash was stopped and the sensory troubles combated with opiates, which required to be administered almost daily during the following three months for the relief of the perpetual pains. Subsequently it was found that she derived more benefit from fifteen-grain doses of antipyrin than any other medicinal agent when the pain was severe, but after use for about a month this drug seemed to lose all its potency and compelled a

return to the opiate treatment after she had been in the asylum eight months.

In the ninth month of her residence with us a new complication presented itself in the shape of gastric crises, and after employing almost the whole armamentarium of medicinal agents without effect, she was found to be fairly relieved by small quantities of iced champagne, together with the application of blisters, the size of a penny, to the skin of the epigastrium.

From this time forward she steadily improved and her mental symptoms sank into abeyance. She was soon able to be up and about again and after a residence of twelve months she became clamorous for discharge. In conversation she showed no change in her delusions and believed that she had been cured owing to the laying on of hands at the prayer-meeting above mentioned. Her physical symptoms were relieved, though the absent tendon-reflexes, ataxic gait, slight inco-ordination and pupillary anomalies, showed that she was only enjoying a remission in the course of the disease. She was allowed to leave the asylum and committed to the care of the Poor Law Guardians. Five months after her discharge she forwarded me a semi-religious publication containing an article written by herself and describing how she had been cured by faith, and a month later a letter was sent to her, in reply to which she practically admitted that her condition remained the same as upon discharge, though still claiming that she was entirely cured.

Some noteworthy points in connection with this case were: firstly, the differential diagnosis from tabetic general paralysis, which was fraught with considerable difficulty in the earlier days of her residence in this institution; secondly, the well-marked remissions of symptoms she enjoyed from time to time; and thirdly, the very definite history of venereal disease. With regard to the differential diagnosis, Gowers, in his "Diseases of the Nervous System," says, "When the symptoms and lesions of tabes are combined with those of general paralysis of the insane, it may be doubtful in which category a case should be placed. The question is rather one of the preponderance of the symptoms of one or the other malady than of absolute distinction between them. In most cases, however, in which this combination exists, the symptoms of general paralysis become more pronounced as time goes on. and

the spinal symptoms, which at first were the most conspicuous, pass into the background."

In the above case the mental symptoms were, at the outset, far more prominent than the physical, and this condition subsequently became reversed, in which state the patient remains. It is now probably between six and seven years since her infection with the specific virus, and the history since her admission covers a period of twenty months.

SOLITARY TUBERCLE OF THE CORD SITUATED AT THE EXIT OF THE SECOND AND THIRD SACRAL ROOTS. *Bulletins et Mémoires de la Société Médicale des Hopitaux de Paris*, No. 10, 1897. By Marfan.

A child of two years and two months was suddenly completely paralyzed in the lower limbs. It was thought at this time that there was complete anæsthesia of these parts. Retention of urine lasted for three days, and was followed by incontinence of urine and feces. After three weeks some movements of the limbs could be made, and walking became possible, but with considerable difficulty. The tendon reflexes were exaggerated, but ankle clonus was not observed. Sensation appeared to be normal. The lower limbs were somewhat atrophied and rigid, and there were some trophic lesions. The upper limbs and face were not affected.

Pott's disease is the most common cause of paralysis in children, but the paralysis does not develop acutely, as in this case. There was also no deviation of the spinal column and no tenderness. Hematomyelia seemed to be the only condition which could explain the symptoms. In a child of two years this might be due to syphilis or tuberculosis. There were no evidences of the former disease in the parents or child. The diagnosis of solitary tubercle of the cord was made, notwithstanding the rarity of the condition, although it occurs more frequently in children. There were no other clinical signs of tuberculosis.

At the autopsy a solitary tubercle of the sacral cord, with hematomyelia of the adjacent parts, subarachnoidal hemorrhage, ascending hemorrhagic poliomyelitis, and intense and diffuse phlebitis of the pial veins was found. No tubercular lesion of more ancient date were observed, although miliary tubercles were present in the lungs.

SPILLER.

A CASE OF "MYOTONIA CONGENITA."

By W. H. HAYNES, M.D.,

Attending Neurologist to the Central Throat Hospital and Polyclinic: Assistant Physician, Neurological Department, Brooklyn Eye and Ear Hospital: Member of the Brooklyn Neurological Society, etc., Brooklyn, N. Y.

The following case is described under the above heading, more on account of the clinical phenomena than for the accuracy of the nomenclature. The patient was presented to the Brooklyn Neurological Society at its meeting in February, 1897, and the opinion expressed by all was that they had never seen a similar case, and were unable to classify it.

Peter C., aged 28, born in Pennsylvania, single, and by occupation a laborer. His father died, aged 98; mother still alive, aged 72. She had six children, two of whom died in infancy. Some of her near relatives have died of phthisis.

He is strongly addicted to alcohol and tobacco, denies, and there is no evidence of, any venereal diseases. When a boy he was in the habit of staying in the water of a pond on his father's farm all day diving and remaining under water for a long time, thereby in one instance nearly losing his life by drowning, and to this habit his people ascribe the source of his present disorder. It was first noticed when he began attending school at the age of seven years. His teachers then observed the difficulty he had in pronouncing words. At fourteen he could protrude his tongue but half-way, so that his parents thought he was tongue-tied and tried to have the phrenum cut, but were informed it would do no good, as it was not the cause of the difficulty. He has never had any illness, with the exception of being laid up for five weeks greatly prostrated after the drowning accident before mentioned, and excepting, at 16 years of age, fracture of both bones of right forearm.

Six or seven years ago, following a debauch, he had an attack of remittent fever and bronchitis, accompanied

by hoarseness, which has continued with increased difficulty of speech, better or worse, according as he had or had not a cold.

Two years later, following an attack of so-called intermittent typhoid fever and grippe, which lasted seven weeks, and which was also preceded by a heavy debauch,

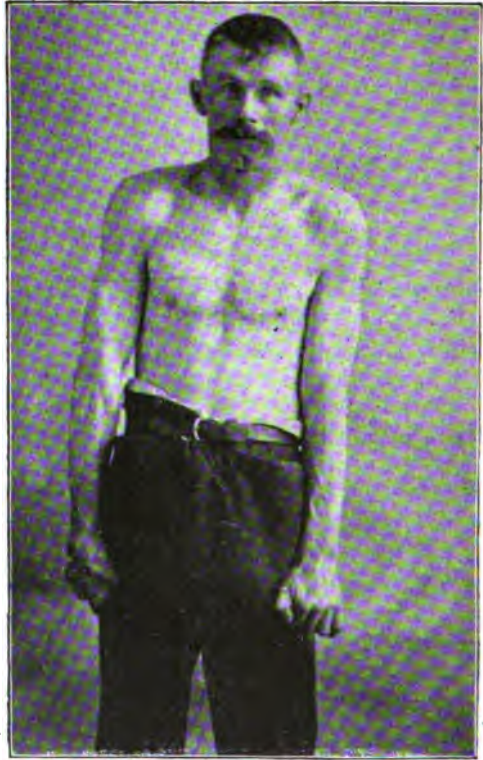


FIG. NO. I.

it was noticed that he was more nervous, and that his arms, noticeably his right, were becoming stiff. He had choking spells while partaking of solid food, two or three times during a meal, and last July or August had one so bad that he nearly choked to death, and could not thereafter swallow for two weeks, when that power suddenly

returned; even now his sister has to cut his food into fine particles so that he may chew and swallow it without danger. During the last four years he has remained *in statu quo*. There is no history of convulsions, and the development has been very slowly progressive.

At present he is not emaciated or anemic, weighs 140

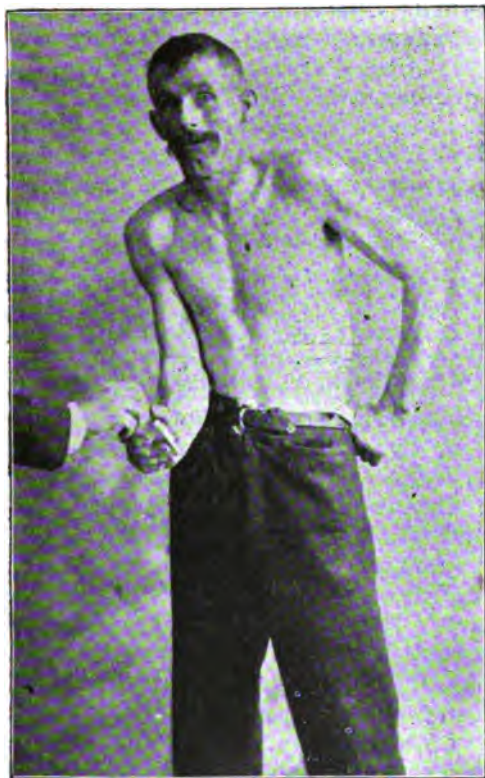


FIG. NO. II.

pounds, and has a good color and appearance. Complains of cough, hoarseness, dyspnoea on exertion, difficulty in swallowing food, "getting stuck in his throat," and pain in the left arm at times; otherwise general health good as regards appetite, walking, working, and feelings generally. Sleeps well.

On examination (Photograph No. I.), it is noticed that

he is restless and has no fever; pulse 80; no heart lesion; respirations, sitting, 22; lips bluish. Memory good, sight and hearing normal, no nystagmus, pupillary, or eye muscle symptoms, optic nerves cloudy, particularly nasal side of left disk. When asked to put out his tongue, it appears as a short, thick, jerking, trembling mass, with general tremors, not fibrillary, running through it. He is unable to protrude it without jerking it in and out. Taste is normal. Speech is of a rapid, nasal, muffled character. He talks in a jerky whisper, as though he were throwing the words out of his throat, and has difficulty in using his for phonating. On inspection, the pharynx and larynx are both seen to be intensely congested and swollen; vocal cords thickened and congested, but not paralyzed. The mucus membrane of the pharynx is perfectly insensible; touching it produces no gagging. The spasmodic condition of the muscles of the lips appears to give rise to the peculiar appearance or expression of the lower part of the face.

The right arm was first affected, and two years later the left. He is right-handed. Hands are unsteady and cannot be kept at rest on account of the marked rigid spastic condition, and general tremor which is most marked in the right, and which is worse when "nervous" or noticed. When standing, arms are seen to hang partly flexed at elbow, and rotated inwards; as a rule, right hand hyper-extended and fingers flexed; left hand, wrist and fingers flexed; some irregular spasm of fingers with a remote resemblance to athetoid movements; no apparent loss of power as shown by work and unbuttoning clothing without difficulty, except for spasms present. Dynamometer, left, 55-45; right 55-55. There is no paralysis.

In taking objects with his right hand, he first rotates the hand and takes the object from the under side, just the reverse of the way an object is usually taken. This appears to be his habitual way, but it is possible for him, with an effort, to take it in the natural way; when asked to do this, that is, take things from above (as we do), not below (see photo. No. II.), he keeps the upper arm close to his side, whereas, when allowed to take them "his way," the elbow is held away from the body, as we naturally do. His grasp in both hands is shown to be very good, and there is no loss of sensibility. Apparently there is no atrophy nor girdle sensation. In the lower extremities nothing is

noticeable except diminished reflexes. No indigestion; bowels irregular; no difficulty in holding or passing the urine.

Of course a number of conditions have been suggested, such as cerebro-glossoplegia or pseudo-bulbar paralysis; so-called Huntington's chorea, para-myo-clonus multiplex, lesions of both hemispheres, etc., but to none of which can this case, on close examination, be set down. The ultimate opinion of those who have given the case the closest study, is that it is probably due to some teratological defect of congenital origin.

NOTE ON A CASE OF ASTASIA-ABASIA.

D. V. Leblond (*Journal de Médecine*, December 25, 1896) reports the case of a girl, nine years old, active, intelligent and without heredity nervous antecedents, who was suddenly attacked with severe pain in the legs, malaria fever and sore throat. The next day an eruption appeared on the legs resembling erythema nodosum, which was followed by slight œdema and diffuse redness. This disappeared with the pain, after rest in bed, and several doses of salicylate of sodium. As the patient was not suffering any more she asked to get up, but on attempting to do so she fell down. There was no vertigo or loss of consciousness, but an absolute impossibility to remain standing or take a step. When in bed all the movements of the lower extremities were preserved. There was no trouble of co-ordination of the upper extremities. A cold douche was followed by hysterical crisis, contraction of upper and lower extremities and loss of consciousness. A second one was better tolerated and later on when attempting to arise after the administration of a douche, the feet did not slip on touching the floor, but she was forced to hold on to the bed to remain standing. Little by little she risked making a step, holding firmly to the bed, and after half an hour's effort she succeeded in walking. This short history is interesting, owing to the mode of onset, in a child undoubtedly impressionable, but without nervous antecedents, and by the absence of hysterical stigmata. At first the diagnosis was difficult. The pain, subcutaneous nodes, œdema and angina, suggested rheumatism and a spinal complication. Without doubt there was absence of hysterical stigmata—no hysterical zone hemianæsthesia or narrowing of visual field—Astasia-abasia can, however, be a mono-symptomatic manifestation of hysteria. Persuasion had no effect on this case, but isolation and separation from the parents during a short sojourn in the hospital sufficed with the douches to produce a positive cure which has persisted now for ten months.

FREEMAN.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 22d, 1897. President, Dr. Charles W. Burr, in the chair.

Dr. Alfred Stengel presented a case of "Arsenical Neuritis," occurring in a child who had been treated for chorea with Fowler's solution.

Dr. Francis X. Dercum.—The gait in this case is extremely interesting. It is like that of multiple neuritis in the adult. I would rather describe it as a "high-stepping gait" than as an ataxic gait. It also looks as though the extensors of the wrist were a little more flaccid than normal.

Another point which suggests itself is, whether or not all other causes than arsenic have been excluded. Is it possible that this has been a grip neuritis, or the result of a combination of several causes as, for instance, grip and arsenical poisoning? The rarity of the case makes one suspect some additional factor.

Dr. Charles K. Mills.—Is it necessary to regard this case as one of arsenical neuritis? Presuming that it is due to the influence of arsenic, may not the toxic effect have been exerted on the spinal centres rather than upon the nerves themselves? The absence of sensory symptoms, perhaps, points in this direction. We could have the train of symptoms presented here in a myelitic affection. This, however, would not alter the prognosis.

Dr. Alfred Stengel.—The condition of the arms was looked into at the time; but I could never demonstrate any loss of power in the extensor group, and I do not believe that there ever was any. I think that possible, coincident, casual factors, particularly influenza, may be excluded, as the patient was in the hospital when the condition developed, and it was in mid-summer, at a time when influenza was not prevalent.

Dr. Charles W. Burr read a paper, entitled: "A Case of Progressive, Neurotic, Muscular Atrophy." (To appear in a later number of this journal.)

Drs. F. X. Dercum and Isaac Leopold read a paper, en-

titled: "A Case of Primary Neurotic Atrophy" (see this journal, page 400.)

Dr. Francis X. Dercum.—I had the opportunity of seeing the case, which Dr. Burr reports, at the Orthopedic Hospital, independently of Dr. Burr, and came to the same conclusion, namely, that it was one of peroneal, muscular atrophy. I referred to this case at the November meeting of the Society. The case reported by Dr. Leopold and myself makes the fifth published in America. Sachs has reported two, Potts one, and Burr one.

Dr. Wm. G. Spiller.—Histologists were beginning to think they knew a little about the pathology of this disease. It has been supposed to be chiefly an affection of the peripheral nerves, although certain investigators have found lesions of the posterior columns and of the anterior horns of the cord. We were not prepared, however, for the interesting report which Oppenheim and Cassirer have recently published. You will notice in this reprint of their paper, that even the terminal nerve branches within the muscles are normal.

In 1893 Dejerine and Sottas published a paper, entitled: "Interstitial, Hypertrophic and Progressive Neuritis of Childhood," in which they presented the description of a disease not known at that time. It has many of the symptoms of primary neurotic atrophy, but in addition there are ataxia, fulgurant pains, nystagmus, myosis with Argyll-Robertson pupils, kyphoscoliosis, and great hypertrophy of the nerve trunks. I am able to present to you specimens of certain of the nerves from one of the cases published by these writers, and you will notice that there are scarcely any normal nerve fibres within the thickened bundles. Within the cord the ordinary degeneration of tabes may be found. Dejerine has recently published another case. There have only been four cases of this disease reported, and during my sojourn at the Salpêtrière I was able, through the kindness of Prof. Dejerine, to study the two patients now living, and the specimens from the case with autopsy previously published. I can assure you that it is a most interesting type of disease.

Dr. F. X. Dercum presented three cases of the family type of cerebral diplegia (see this journal, page 396).

Dr. Charles K. Mills.—These cases are similar to those published by me three or four years ago in one of the reports of the New Jersey Training School for Feeble-Minded Children. I referred to two or three groups of cases found there, in which other members of the family had similar trouble.

Dr. Wharton Sinkler.—Some time ago, I had a group of three cases in the same family, in which Dr. Osler took much interest. He regarded them as cases of Friedreich's ataxia. I was in doubt at the time, whether or not they should be

classed under that head, not only on account of the spastic condition which existed, but also because there were many symptoms of a distinctly cerebral origin. There was evidence of cerebral disease plus cord disease, although, apparently, the cord symptoms had been the earlier. There were four cases with the same symptoms in the family, but I saw only three of them.

Dr. John K. Mitchell.—I have notes of two cases of a paraplegic type without mental affection. Both occurred at an early age, and both followed slight attacks of acute disease. The disease began in the second year, and both have since had advanced spastic paraplegia. They are now sixteen and eighteen years of age. There were eleven children in the family, all of the others being in good health, with the exception of one who has epileptic attacks.

Dr. J. P. C. Griffith.—I would like to ask Dr. Dercum, whether he would not distinguish between heredity, or family, spastic paraplegia and Little's disease? He speaks of Van Gehuchten as discussing the latter, but, as I understand the articles, the recent writings of this author discuss not *hereditary* spastic paralysis, but *congenital* spastic rigidity or Little's disease. Sachs rightly, I think, claims that the two are to be sharply differentiated. Van Gehuchten was not considering at all such cases as Dr. Dercum reports. He goes even farther and limits Little's disease to the cases in which not merely an accident during birth, but a developmental defect, the result of premature birth, is also operative. Hereditary, spastic paraplegia very commonly first shows itself later in life. It is not congenital, as Little's disease is.

Dr. Dercum has not mentioned the interesting cases of the family form of spastic paraplegia reported by Neumark in 1893. I notice he has recently made a further contribution to the subject.

I have now under observation two children which strongly suggest the presence of hereditary spastic paralysis, but I have not yet studied them sufficiently to be able to publish them as such.

Dr. John K. Mitchell.—To say that this is or is not a congenital condition is merely a question of terms. The nervous systems of these children are, as it were, hung on hair triggers, and a very slight cause is capable of exciting great disturbance. After the first symptoms are seen, this disease develops too rapidly to be a degenerative disease without previous disorder. Some primary deficiency must necessarily be at the bottom of a trouble which appears so rapidly and starts from such trifling causes.

Dr. M. V. Ball.—I wish to report two cases. The older child, aged seven years, is a congenital idiot with a spastic con-

dition. The younger child is feeble mentally, and we may look for a spastic condition in its case. It does not yet walk. I believe that it is not uncommon to find several congenital idiots in the same family with a spastic condition in some of them.

Dr. F. X. Dercum.—Cerebral diplegia arises from two very different causes, first, cerebral diplegia may be due to imperfect or feeble development of the neurons of the motor area. Here spontaneous degeneration may lead to the appearance of spastic palsy, or the vitality of the neurons may be so low that they perish from very slight causes, for example, the infection of measles. Secondly, there is diplegia due to trauma received during birth (dystocia). To this group the term "Little's disease" should be restricted. It may occur in children whose nervous system presents a normal degree of resistance.

Dr. David Riesman presented two cases of brachial tremor. The first patient, a married woman, aged 54, has had a fixed torticollis for 10 years, which she attributes to sleeping one night in a draught after having washed her hair. Forty years ago she suffered from an attack of rheumatism, and has been more or less rheumatic from that time. Since January, 1896, she has had a rapid rhythmic tremor of the right forearm, ceasing during voluntary movements and during sleep. The movements are 6 to the second. There are no tremors in other parts of the body; the knee jerks are exaggerated; ankle clonus is not present, and sensation is normal.

The patient states that three months before the onset of the affection she saw a man in a car who was suffering from marked tremor; she was greatly startled, and the sight made a strong impression on her mind. Before the onset of her trouble she was also subjected to great mental strain by her husband's intemperate life.

The speaker considered the case probably one of hysteric tremor.

The second case was a married woman, 40 years old, who for 14 years had had a violent spasm of the right arm. It came on after her second confinement, during the lying-in. She was frightened by a fire in the neighborhood, and for five days had generalized spasms of all four extremities. These movements subsided in all the limbs except the right arm. For the last six years the movements have stopped scarcely more than 15 minutes at a time, when the patient was in repose, though they always cease during work. Excitement greatly intensifies them. She suffers also from palpitation, and on examination was found to have a double aortic murmur.

The movements of the arm are of large amplitude, and when the hand, during the spasm, is resting on any surface, a loud, patting noise is produced.

On account of fugitive rheumatic pains, the patient was at

first placed on a mixture of potassium iodid and sodium salicylate. She improved greatly, and on her third visit the tremor had ceased, and since then, September 15th, 1896, she considers herself cured, as far as the tremor is concerned. Considering that the affection had lasted 14 years, and had been constant for the past 6 years, it can only have been hysteric in nature, the recovery being attributable to the influence of suggestion.

Dr. F. X. Dercum.—The first case would appear to be one of paralysis agitans, though somewhat atypical. There is rigidity of the back of the neck, slowness of movement and fine tremor, arrested by volition. The tremor is not typical in distribution, but this, as is well known, varies greatly. Festination is not always present in paralysis agitans.

Dr. Wharton Sinkler.—I agree with Dr. Dercum that this is probably a case of paralysis agitans. I had a case in an elderly man who had been subjected to a severe shock by seeing a falling elevator shoot past him as he stood by an opening. Tremor began at once in the right arm and never ceased afterward except under voluntary effort. Many other instances are on record of paralysis agitans taking its origin in sudden mental impressions, such as fright, grief, or injury.

Dr. D. Riesman.—I do not think that the condition of the neck can be taken into account as sustaining a diagnosis of paralysis agitans. The neck has been this way for ten years. There has been no complaint of sweats or vasomotor symptoms. While I cannot bring any other arguments against the diagnosis of paralysis agitans, yet time will settle the question.

Dr. Wm. G. Spiller presented an unusual case of hemiplegia (see this journal, page 391).

Dr. Charles K. Mills.—It is not probable that this case is one of hysteria. I believe that the man is suffering from a hemiplegia of organic origin, although he may have hysterical symptoms, but these are epiphenomena. He may have had a hemorrhage, not very large, into a certain part of the internal capsule. The paralysis of his arm and leg is to my mind that so often seen in organic hemiplegia; so also is the tremor, which is like that which has been described as hemiplegic or post-hemiplegic tremor. It will be noticed that he has some facial hemiparesis, although this is not very marked. The contractions in arm and leg do not seem to be of the hysterical type.

Most conclusive of all to me is the foot clonus which is shown by this patient; it is the clonus of organic hemiplegia. Of this I feel certain from many years' experience in the examination of hysterical and organic cases, and I would like to strongly emphasize the importance of the symptom ankle clonus in the differentiation of hysteria from organic disease. While the knee jerk may be, and frequently is, excessive in

cases of grave hysteria of the hemiplegic or of other types, I have never yet seen a case in which the persistent clonus, which is so significant of certain forms of organic disease both of the brain and spinal cord, was present. Occasionally what might be termed a *pseudoclonus* is observed. Gowers has described this as a spurious clonus, and believes that it is due to a half voluntary contraction of the calf muscles.

In the vast majority of cases of hystero-traumatism, of which I have examined a large number, and especially during the last four years, even this form of spurious clonus is usually absent. It is misleading for writers on hysteria to speak of the presence of ankle clonus in such a way as to give the impression that it may be of a well-marked and persistent character like that which is seen in organic disease. Several important matters must be borne in mind in this connection, as, for instance, the fact that organic disease, either spinal or cerebral, is often associated with hysteria or hysteria with organic disease, and the presence of a true clonus in such cases may have misled the diagnostician. I have seen a few cases of this kind: one, for instance, in which the persistent ankle clonus was present, and in which the diagnosis of hysteria was made and adhered to for some time, but the case eventually proved to be one of organic spinal disease, probably lateral sclerosis. In another case, which fell under my observation, and which has been cited by one of my colleagues as proving that ankle clonus is sometimes present in grave hysteria, it was believed that the patient had angular curvature with compression of the cord. It was supposed that she could not walk, and for a long time she had been in a wheeling chair. On one occasion, after the administration of some remedy, practically a placebo, she got up and walked. This case was, I believe, one of partially cured caries and local pachymeningitis. Although the patient had partially recovered, the cord at a certain level was probably still subjected to some irritation and possibly compression, the latter not being sufficient to destroy its transmitting functions. This case may have been one of hysteria, but the hysteria was probably imposed upon an organic disease. Ankle clonus of the genuine persistent type was present, but I do not believe that it proves anything with regard to the significance of the symptom in the diagnosis of hysteria.

Dr. James Hendrie Lloyd.—Some time ago I was called in consultation in this case. The rather unusual question had arisen, whether it was desirable, on account of the extreme tremor, to amputate the arm. Amputation of the arm for post-hemiplegic tremor was not a procedure which I could endorse, and the project was finally abandoned. I demonstrated that, the trouble being of central origin, amputation of the arm would remove only part of the structures that were affected,

and was entirely unjustifiable. The demand for amputation, it seems, had been made by the patient himself, who declared that he would commit suicide if something were not done for his relief.

When I examined the man a year ago, I had no hesitation in making a diagnosis of organic lesion of the brain, and I said then that this tremor was only a grave type of posthemiplegic tremor. I still think that that is the correct diagnosis. I am ready to believe, however, that there may be some hysterical stigmata complicating the case, as is not unheard of.

I would call attention to the fact that the history of hemianesthesia, paralysis of all four extremities, and amnesia for languages, which were said to be present in the early stages of the case, is to a large extent based upon hearsay evidence. We have no very authentic data as to the man's condition at the commencement of the attack. I wish to go on record as having expressed the opinion a year and a half ago, that the man had organic hemiplegia. I base this diagnosis upon the type of the hemiplegia, which is organic rather than hysterical; *i. e.*, it is worse in the arm than in the leg, and is accompanied with characteristic and exaggerated reflexes (especially a well-marked ankle clonus); that the tremor is unilateral and confined to the affected side; that there is not a true hysterical hemianesthesia with involvement of mucous surfaces; and, finally, that there is no contraction of the visual fields satisfactorily demonstrated. If this man has not a hemiplegia of organic origin, then I do not see how henceforth we are to distinguish organic hemiplegia.

Dr. F. X. Dercum.—I regard this case as unequivocally one of traumatic hysteria. The fact that he had hemianesthesia is very suggestive; also the time that elapsed before the onset of the symptoms. It is not probable that he had a hemorrhage so long after the accident. I have seen ankle clonus in traumatic hysteria, and have records of such cases. In my mind the presence of an ankle clonus militates in no wise against the diagnosis of hysteria.

Dr. Wharton Sinkler.—The case seems to me one of traumatic hysteria for the following reasons: The man was totally paralyzed; he was in a condition of inhibition of voluntary function, so that, although he could hear sounds, he was unable to raise himself from the floor or to call for help. The aphonia is also typical of hysteria and is quite unlike organic brain disease. The history does not belong to that of any form of hemiplegia from organic disease. I am certain that ankle clonus may exist in hysteria, particularly in traumatic hysteria.

Dr. A. Ferree Witmer.—The involvement of the fibres of the seventh nerve is of extreme interest. I should like to mention a similar condition that I saw in a young woman in the

eighth month of pregnancy, in whom there was evident facial palsy. There had been a marked element of hysteria in the family for three generations. I regard this as a case of hysterical facial palsy, due to the excitement attending her condition.

Dr. F. Savary Pearce.—In regard to the diagnosis of organic disease in this man, I can cite a case of severe muscular strain producing hemorrhage from the lateral sinus, which was seen at the autopsy. This man presented to-night was also subjected to great muscular effort. In Dr. S. Weir Mitchell's practice I have recently seen a woman of 56 years, who in March, 1896, had a hemorrhage involving the speech centre, from which she slowly recovered. She was conscious during the onset of the attack and could speak. The patient had right hemiplegia. I mention this case as an evidence that there may be organic paralysis without mental disturbance.

Dr. Wm. G. Spiller.—I am sorry that no one has spoken of the peculiar disturbance of speech. The patient lost his power of speaking in all languages except one. I can recall a similar case which I saw in the clinic of Krafft-Ebing in Vienna. The diagnosis between hysteria and organic disease in that case was not easy. Such a partial loss of languages may, of course, be organic.

I do not think we should regard the predominance of the contracture in the upper extremity necessarily as due to organic disease. It must be borne in mind that the man is, and has been, very excitable; and that he attempted to take his life a short time ago. A few years ago he had a tremor in the right lower limb very similar to that now seen in the upper. This has entirely disappeared, except when the patient is excited. The tremor in the upper limb is, to my eye, characteristic of hysteria, so much so that the diagnosis of hysteria presented itself to my mind the instant I saw the man. True ankle clonus is uncommon in hysteria, but Gowers does not deny its existence. He says, it is "extremely rare." I have presented this case as an unusual form of hemiplegia. Shall we make ankle clonus a test for organic disease? I would hesitate to call every case organic in which it might be found. The severe mental strain, the late appearance and mode of development of the hemiplegia are in favor of the diagnosis of hysteria. The man is very intelligent, and I see no reason for doubting the account which he gives of his disease. His statements in no way conflict. We must remember that certain stigmata of hysteria may disappear after a number of years. The slight disturbance of consciousness during the first hours of the paralysis, the ability to notice everything about him, even to recall the hour, the aphonia, the complete paralysis of all four limbs form a combination of symptoms more like that of hysteria than of organic disease. Shall we disregard the clinical

history in a given case, simply because it does not agree with our views? On this the diagnosis in the present case must chiefly rest.

Dr. James Hendrie Lloyd reported a case of cerebellar tumor and presented the specimen.

Dr. H. A. Hare read a paper, entitled: "A Report of a Case of unusual Edema in Hemiplegia. (To appear in a later number of this journal.)

Dr. James H. Lloyd.—I would like to ask Dr. Hare if he took into account the possibility of this being a hysterical edema. We sometimes have remarkable examples of edema in hysteria. I saw a marked instance of this in a medico-legal case of brachial monoplegia due to an electric shock. The arm was flaccid and presented an extraordinary blue edema, such as has been described by Charcot.

Dr. F. Savary Pearce.—May not the mitral disease account in part for the edema in the paralyzed arm where all function is sluggish? Dr. Hare tells me that the patient at one time had edema of other parts.

Dr. H. A. Hare.—This was not a blue edema.

ANALGESIA OF THE ULNAR NERVE IN THE INSANE.

Dr. Arrigo Giannone gives a detailed account of his investigation regarding the condition of the ulnar nerve, in July No. of *Rivista di Patologia Nervosa e Nuntale*; this work was investigated by the researches of Biernachi, who found analgesia of this nerve in locomotor ataxia.

In 47 cases of Dementia Paralytica, there was analgesia of both nerves in 21 cases (males); analgesia of one side in 2 cases (males); there was lessened sensibility in 10 cases (males); normal in 6 cases (males); analgesia in 2 cases (females); lessened sensibility in 2 cases (females); normal in 4 cases (females); recapitulating, the ulnar symptom was found in 53.2 per cent, lessened sensibility in 25.6 per cent., and the nerve was normal in 21.2 per cent.

In an examination of 210 insane, the ulnar symptom was found in 53, or 24.9 per cent.; lessened sensibility in 53, or 24.9 per cent.; normal in 107, or 50.9 per cent. Similar researches were made in regard to patients with locomotor ataxia with the following results: Out of 15 patients the ulnar symptom was present in 9, or 60 per cent.; lessened sensibility in 2, or 13.3 per cent; normal in 4, or 26.7 per cent.

In regard to its importance in locomotor ataxia the author concludes:

1. That the analgesia of the ulnar nerve is not to be considered in the same category as the other symptoms of locomotor ataxia.

2. That considered by itself, it is of little importance, being met with much less frequency than the Argyll-Robertson pupil or Westphal's symptom.

3. That it is not pathognomonic of locomotor ataxia, but occurs in other diseases of the brain and nervous system. KRAUSS.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, March 2, 1897.

B. Sachs, M.D., President in the chair.

REMARKS ON THE MORBID ANATOMY (CHIEFLY OF THE BRAIN AXIS) IN DEMENTIA FROM LUES.

Dr. E. C. Spitzka read a paper with this title. He said that while cerebral syphilis was recognized clinically in most cases by a certain uniformity of type, it was still so variable in its manifestations that each case must be regarded as a law unto itself. Cerebral syphilis being chiefly an arterial disease, and the nutrient vessels being chiefly terminal vessels, each thrombic or gummy arterial lesion constituted almost an atrophy experiment. In this disease, epithelial granulations are extremely common, and are of two varieties, viz.: (1) Round, small, regularly distributed and almost translucent; and (2) warty, firm, opaque and interlacing. The latter are the older and contain connective tissue elements.

Dr. Spitzka then quoted the following case: J. R., forty-seven years of age, married nineteen years, had been seen by him on January 21, 1881. The patient complained of weakness of the limbs and of fugitive pains in various parts of the body. On rising from bed these were better, and they disappeared after walking. There were reflex iridoplegia and pain; the knee-jerk was slightly more jerky than normal, but no more exaggerated; there was no Romberg symptom. The ocular background was normal. There was right ptosis of extreme degree. The pupils were unequal. All the associate movements of the eyeball were normal. There was no marked tremor of the hands. The tongue was protruded steadily in the median line. There was no facial paresis. The urine was normal, and there were no bladder symptoms at any time. He was seen at intervals during twenty months. At no time was it possible for him to remember facts when recalled to him by association, and at no time was he unable to attend to the details of his business, yet all his acts impressed both lay and professional observers as automatic. His family noted an exaggerated irritability. Mixed treatment was prescribed, but was not carried out regularly. Four months after the time when he was first seen he had an apoplectic seizure, which was followed by an insufficiency of the left rectus muscle. A second and similar seizure was followed by left ptosis. After the first attack mercurial inunction caused prompt improvement,

but not so after the second seizure. Two days before death the speaker had seen him. For some days previously he had been constantly making the tour of the house, and when seen was busily engaged in ripping the binding from the furniture. He went into coma a few hours later and died in this state. About this time his baby came under observation with a distinct papular syphilide. At the autopsy, the dura and arachnoid were entirely normal. The vessels of the convexity were apparently normal. The basilar artery was slightly thickened in its anterior third, and the right posterior cerebral showed two yellow patches. The left ventricles were normal. The slope of the aqueduct of the third ventricle was filled with net-like and firm granulations. The posterior division of the fourth ventricle contained numerous warty granulations. The brain axis was preserved according to the methods then in vogue. Two sections were exhibited—i. e., from the anterior part of the corpora quadrigemina and from the crura. The chief nuclear changes in this case were limited to the posterior division of the pneumogastric nucleus.

This case was contrasted with the following, which exhibited a typical picture of paretic dementia. The syphilitic infection had occurred twelve years before death, but the mental disorder had lasted only fourteen months. Death occurred in an apoplectiform sequel of a delirious exacerbation. The fore-brain showed no marked lesion of the neural elements; there was, however, a marked proliferation of the "spider cells." In the brain axis were diffused proliferations of nuclear elements in the reticular formation of the pons, and there was actual cirrhosis in the raphe and course of the hypoglossal. The speaker said that the difference of the location of the arterial lesion in the brain axis in cerebral syphilis determined whether the case was to be one of syphilitic dementia or paretic dementia. With the intensification of the process in the aqueduct of the former was to be expected. The vessels of the reticular formation are not strictly terminal, and nutritive changes are apt to be followed by insidious and irritative damage to the tissues, and corresponding influences on the function and tracts there located.

The President said he had been much interested in these comparatively little known findings in the lower portion of the brain axis. For the past few years he had been struck by a number of cases which, clinically, had the appearance of general paresis, but which, in their general course and development, proved to be entirely atypical. Then there was a class of cases which from the outset appeared to be distinctly syphilitic, because associated with the mental symptoms were the other symptoms known to be decidedly syphilitic—e. g., oculomotor palsies and frequent epileptiform seizures. He had

taken some pains to determine clinically the symptoms which would enable us to differentiate between the typical paresis and the cases which yield a more favorable prognosis.

Dr. Sachs then referred particularly to a gentleman who had been stricken about one and a half years ago with very marked delusions of grandeur and had become entirely unable to take care of himself and family, who were travelling with him at the time. His memory forsook him and he was compelled to choose between confinement abroad or an immediate return to this country. A few days after his return, Dr. Sachs had seen him. At this time he had presented every single symptom of parietic dementia. There was the characteristic speech and the immobility of the pupils; he was unable to write his name at that time, etc. These symptoms gradually increased. He was placed under the care of an able trained nurse and for about six months there was no decided change. After this time a gradual improvement was noted, and at present—eight months later—the man is entirely well, and is able to attend to his business. The only remaining symptoms are complete immobility of the pupils and absence of the knee-jerks. His mental condition is now entirely normal. This patient had confessed to a syphilitic infection many years ago. He had two healthy children and there was no history of miscarriages. This case, Dr. Sachs said, seemed to him to present a very different clinical course from that of ordinary general paresis. Apparently the specific meningo-encephalitis might be recovered from. In the other cases that he had seen he had looked upon the ultimate prognosis as grave, but in the one just cited he was inclined to believe that the outlook was much more favorable.

Dr. W. M. Leszynsky said that the experience of most observers with cases of parietic dementia was that there were remissions simulating recovery. During these remissions the improvement was so marked that the patient was often discharged from an asylum as "cured." Usually, however, there would be a recurrence within a year.

Dr. W. Granger said that remissions in cases of paresis would be observed in both the syphilitic and non-syphilitic. The case cited by Dr. Sachs was very different from those in which remissions were usually observed; the almost complete restoration of the mental condition in this case would seem to indicate the possibility of ultimate recovery. Remissions in paresis would not be found to be perfect if the conditions were thoroughly and searchingly investigated. The power of concentration, of continued work and of self-control in the annoyances of daily life, would be found defective, showing that there was not a full restoration of the mental integrity. It would be remembered that for many years Dr. Henry Hurd, a

careful observer, had reported what was thought to be a cured case of general paresis. In the report of this case, however, it was noted that the man did not have his former energy and power of attention to details. After a remission of many years there was a recurrence and death ensued.

The President said that there was another order of cases of general paresis suggesting the possibility of the clinical symptoms being due to a true syphilitic morbid process and not to the ordinary morbid process of general paresis. This view had been suggested to him by the case of a gentleman who had been seen also by Dr. Spitzka about ten years ago. At that time there had not been the slightest doubt of the existence of general paresis. The man had been in a condition of dementia, however, for six or eight years. The disease developed very soon after the initial lesion of syphilis. In this case it seemed to him that the dementia was due to a widespread meningo-encephalitis, with resulting sclerotic changes.

Dr. Spitzka said that cases like the one to which Dr. Sachs last referred, of the continuance of life without restoration of mental power, where the typical symptoms of parietic dementia were present, were far from rare; there were at least a dozen illustrative cases now residing in this city. He had seen distinct remissions lasting fully four years and a large number lasting for a year and a half. He would, therefore, wait at least two years before feeling sanguine about the case referred to by Dr. Sachs. The persistence of the Argyll-Robertson pupil, or of the absence of the knee-jerk was not, however, an obstacle to recovery, because they were the last to disappear—indeed, the condition might have existed before the case came under medical observation or there was evidence of any disease. In the series of cases specially considered in the paper, the lesions occurring in the very locality where one would expect the most serious, if not fatal, consequences, had yielded the best prognosis. One of these cases had been seen by Dr. Seguin and his staff, sixteen years ago, when the man was in a condition of coma justifying the statement that he was moribund. Another one had been seen by one member of this society who had pronounced him dead nine years ago. In both cases, glycosuria with a large percentage of sugar had persisted to this day. In the first case there was only one mental symptom, viz.: when he shut his eyes he lost the sense of his identity. The other patient presented another singular anomaly, i. e., he could remember every fact that had been brought before him up to the time of his comatose or apoplectic seizure with an accuracy that was very remarkable, and yet five seconds after he had answered such questions he could not tell what questions had just been propounded to him. Dr. Spitzka said he had been puzzled to account for the fact that

all past registration should be intact in a man fifty-three years of age and yet his judgment should be so little embarrassed that he should make application for the appointment of a commission to take care of his business. If there were a degenerative process in the cortex it should have shown itself in eleven years by an abolition of recollections, or at least by an abolition of associations, or by impairment of one's sense of identity, yet none of these things had occurred.

Regarding the effect of anti-syphilitic treatment on these conditions, the speaker said that as long as in these cases there was improvement, or prolongation of life, or an increase in the number and length of the remissions following such treatment, he would hold the door of hope open regarding the curability of dementia. Those who argued that if cases of supposed parietic dementia recovered they could not be examples of parietic dementia, belonged to another planet.

REPORT OF A FATAL CASE OF HEMORRHAGE INTO THE PONS.

Dr. W. M. Leszynsky reported the following case: At 8 A. M. on January 23d, he had been called to see Mrs. X—, fifty-five years of age, the mother of six grown-up children. It was stated that she had been in excellent health previously. Half an hour before his arrival, and after having taken a cup of coffee, she suddenly lost power on the left side and became dizzy. When seen by him consciousness was preserved; pulse 86 and of high tension; respiration 24 and regular; temperature 80 F. in the mouth. The heart's action was forcible and indicated well marked cardiac hypertrophy without valvular disease. The lower facial muscles were paralyzed on the right side; on the left, the lower facial muscles were in tonic spasm. Both pupils were equally contracted to about 1.5 mm., and were absolutely immovable. She could not voluntarily elevate the left upper eyelid, but could close the right eye. The left eyeball deviated outward; the right was in parallelism. The tongue was protruded in the median line. The left upper extremity was paralyzed. Sense and motility were normal on the right side; the left knee-jerk was feeble. The patient insisted that the vertigo, hemiplegia and inability to open the left eye occurred simultaneously. For the next hour and a half Dr. Leszynsky remained at the bedside and there was no sign of mental hebetude or any perceptible change in the symptoms. The urine contained a large quantity of albumen and many granular casts and broken-down red blood corpuscles. When seen again at 1 P. M., she was completely comatose and in a cold perspiration; there was complete relaxation of the para-

lyzed extremities. The right forearm was flexed upon the forearm very rigidly. Both knee-jerks were exaggerated. Pulse 84, respiration 24 and temperature 99.2. Venesection was suggested, but was delayed until 6 P. M., owing to the absence of the family physician. There was then complete relaxation on the right side and the temperature was 103.4. Sixteen ounces of blood were taken from the right median basilic vein, and the stream of blood indicated great pressure. At midnight, pulse 120, respiration 40, temperature 101.8 in the axilla. At 4 A. M. the temperature was 105 and death occurred at 6.30 A. M., with a temperature of 107. No autopsy was permitted.

Dr. Leszynsky said that he thought it was questionable whether free venesection could have saved her, even though it had been undertaken very early. It was apparently one of those cases of chronic interstitial nephritis with pronounced cardiac hypertrophy without special symptoms until cerebral hemorrhage unexpectedly terminated life.

Periscope.

With the Assistance of the Following Collaborators:

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NEUROPATHOLOGY.

LABIO-GLOSSO-LARYNGEAL PARALYSIS OF CEREBRAL ORIGIN.

Prof. Picot and Dr. Hobbs (Medical Week, Sept. 4, 1896), reported at the French Congress of Internal Medicine a complete study of a case of this trouble. The patient, 66 years of age, had had several apoplectic strokes followed by right or left paralysis. Three months before his admission to the hospital he suddenly developed paralysis of the lips, tongue, pharynx and larynx, complete except for the velum palati and masticator muscles. Pharyngeal reflex was abolished, masseteric reflex exaggerated, electric reactions normal. The patient died five months after the onset. Small cystic tumors were found over the first right frontal convolution at the level of the isthmus which connects the first frontal with the ascending frontal, and over the first right temporal convolution in front of the Sylvian fissure; also on the left side at about the middle of both the ascending frontal and parietal convolutions. There were slight losses of substance due to old foci of hemorrhage in the caudate nucleus in the right hemisphere; the outer third of the external capsule was destroyed. The medulla and pons appeared to be normal.

MITCHELL.

CASUISTISCHE MITTHEILUNGEN AUS DER HEIDELBERGER MED. KLINIK (Clinical Communications from the Heidelberg Medical Clinic).

Prof. Erb. Deutsche Zeitschrift für Nervenheilkunde, Band 9, Heft 3 u. 4. By J. Hoffmann.

The first of these communications is in regard to a chronic case of bulbar paralysis, in which lead may have been a cause. The disease began clinically with spontaneous tremor of the lower jaw. The chin-jerk was exaggerated. This Hoffmann has frequently noticed in chronic bulbar paralysis.

The second communication is in regard to contra-lateral, electrical reflex contraction in a case of left-sided (cortical?) facial paralysis. A positive diagnosis of the location of the lesion was impossible. Electrical irritation of the right side of the face with a feeble current caused contra-lateral contraction, even when there was no contraction to be seen on the right side of the face. The contra-lateral reaction was not obtained from a feeble current applied to the left side of the face.

Hoffmann reports three cases of occupation paresis in the lower limbs due to cramped position. In all three cases the muscles supplied by the left peroneal and posterior tibial nerves were affected, although the muscles of the right leg did not always escape. The cause was probably pressure upon the nerves.

A case of Thomsen's disease is reported in which atrophy of the flexors of the forearms and of the small hand muscles was noticed. This was attributed to neuritis. Hoffmann regards the disease as a primary myopathy (Erb, Dejerine and Sottas).

Hoffmann relates the history of a case in which tetany developed three days after almost total removal of the thyroid gland. In three weeks the symptoms were much improved. After three months, however, they returned with indications of myxœdema. Myotonic reaction was noted in the muscles. The contractions of the muscles to voluntary innervation, electrical or mechanical irritation, was like that seen in Thomsen's disease, except that they did not yield even after a number of voluntary movements. The tetany, Thomsen's disease and myxœdema in this case, Hoffmann regards as the results of removal of the thyroid gland. The patient's condition was better in warmer weather. Marked improvement was obtained by giving thyreoidin, at first 0.1 daily.

Hoffmann reports a second case of tetany following almost total extirpation of the thyroid gland.

SPILLER.

A CASE OF LESION OF THE TRACTUS OPTICUS AND OF THE PEDUNCLE.
By Dr. A. Mahaim. *Journal de Neurologie et d' Hypnologie*,
No. 10.

The case concerns a man of 21 years with a marked family history of tuberculosis on both sides.

Three years ago the patient first noticed that his sight got worse; this weakness of sight increased gradually more and more. In summer 1894 began to have difficulty in flexing the fingers of his right hand; at the same time the right leg felt heavy and could not be moved as well as the left. This hemiparesis also increased. On January 20, 1896, suddenly taken with violent headache, diffuse, more intense in front than in the occiput; on the same day vomiting several times. On January 21, in a soporose condition.

After temporary remission these symptoms returned with full intensity for one week, then after a new remission, again for three days; but never loss of consciousness. The 13th of February, able to leave the room, sight was very weak, had diplopia which disappeared in a few days, the right arm and leg were contractured and could be moved only with difficulty.

From February until the end March had again—about once a week—attacks of headache with vomitings and somnolence lasting several hours. These attacks gradually lessened in intensity. At that time M. found the following:

Stat. præ: Hemiplegia with contractures, on right side. Right homonymous hemianopsia, reaching to median meridians in both eyes. Visions 5-5 and 5-6 respectively. There is hardly any pupillary response when the light falls only on the left halves of the retina. Deviations of tongue to the right. Otherwise, aside from the right facial paresis, no disturbances in the innervations of the cranial nerves. Sensation normal everywhere.

M. makes the diagnosis of a lesion, probably tubercle of the basis, compressing first the left optic tract, then the peduncle. The points which speak in favor of such localization are the hemiopic pupillary reaction with right hemianopsia, the hemiplegia, or rather hemiparesis, and absence of sensory disturbances, especially of semi-anesthesia.

ONUF.

CLINICAL NEUROLOGY.

THE DIAGNOSIS OF HEADACHES. By Howell T. Pershing, M.D., Colorado Medical Journal, Vol. II., No. 10.

Pershing has analyzed the symptoms of the diseases in which headache is liable to be a prominent feature, and has arranged them in a table so that by comparing the symptoms of an individual case with the headings and sub-headings of the table, the probable diagnosis can be quickly found.

Headache.—Pain in the head not limited to the distribution of particular nerves.

I. There is organic disease of the brain or its membranes, shown not merely by the severity and persistence of the headache, but also by the occurrence of some more positive signs, e.g., paralysis of ocular muscles, face, optic neuritis or optic atrophy; Jacksonian epilepsy, etc. If delirium or stupor occurs, the headache still continues. Vomiting common.

(a) Onset sudden or very rapid, following an injury or occurring in an aged or insane person without evidence of infection. Convulsions, followed by paralysis on side opposite the headache. Sopor and coma with slowness of pulse supreme.—Meningeal hemorrhage.

(b) Onset gradual but acute, with fever. A source of intracranial infection or irritation present. Whole course short. General hyperæsthesia common in early stage. Spasm of neck, general convulsions and cranial nerve symptoms, such as ptosis, strabismus, etc. Optic neuritis occasional. Temperature irregular.—Meningitis.

(c) Onset rapid or slow. Source of purulent infection. Temperature irregular. There may be rigors followed by fever and sweating.—Abscess.

(d) Onset slow. Optic neuritis. Temperature usually normal. No signs of suppuration.—Tumor.

(e) Onset slow, without fever and without intense optic neuritis or slow pulse. Patient alcoholic or syphilitic. Organic disease indicated by spasm or paralysis in the domain of cranial nerves, rarely in limbs.—Chronic meningitis.

II. Positive signs of organic disease of the brain or membranes absent, but there is evidence that the blood vessels are diseased.

(a) Patient is past forty. The arteries are atheromatous. Heart often hypertrophied, and albumen is usually found in the urine.—Arterio-sclerosis.

(b) Age less than forty. Headache chiefly nocturnal and accompanied by insomnia. Syphilitic history.—Syphilitic endarteritis.

III. No organic disease of brain membranes or vessels.

(a) Headache periodic. Pain unilateral, accompanied by nausea and intolerance of light or noise. Family history of similar attacks or of epilepsy or other neuroses.—Migraine.

IV. No distinct periodicity. Pain not unilateral. Headache appears at same time as fever, and disappears if delirium supervenes. There is evidence of general infection without any unequivocal sign of organic disease of the brain or its membranes.—Infectious fever.

V. There is no infection, but there is evidence of a toxic substance active in the system.

(a) Urine contains sugar. Patient suffers from polymia, thirst, weakness, etc.—Diabetes.

(b) Urine contains albumen, or casts, or both. Pallor and œdema often manifest. There may be a characteristic retinitis or optic neuritis.—Uræmia.

(c) Urine free from albumen and sugar. Headache worse in early morning and tends to wear off during the day. Uric acid, diathesis or gouty deposits.—Gout.

(d) History of excessive consumption of alcohol. There may be initiative dyspepsia, insomnia, tremor, etc.—Alcoholism.

(e) Perverted digestion. Headache dull, comes on soon after eating, or is associated with the eating of some particular food, and is temporarily relieved by purgatives and intestinal antiseptics.—Indigestion.

(f) Patient exposed to lead. There may be a blue line on the gums, colic with constipation or bilateral wrist drop.—Plumbism.

VI. No infective or toxic cause but the cerebral circulation is disturbed.

(a) Headache throbbing follows excitement or exertion, and may be associated with flushed face. Increased by coughing, straining or lowering head.—Active Hyperæmia.

(b) Headache dull and heavy. Return of venous blood obstructed.—Passive Hyperæmia.

(c) Heart action weak or blood impoverished.—Anæmia.

VII. No infective, toxic or circulatory cause, but there is a reflex disturbance.

(a) Pain associated with the use of the eyes for near work, and is felt in the brow or back of the eyes, or appears to extend from the eyes to the back of neck. An error of refraction is present or weakness of one or more of the extra ocular muscles.—Eye Strain.

(b) Pain associated with disease of the nose or its adjacent sinuses. Inner wall of orbit may be tender or headache markedly increased by touching the middle turbinated bone with a probe.—Nasal Headache.

VIII. No organic, toxic, reflex or circulatory cause apparent.

(a) Nerve force exhausted from any cause. Sensations in head may be disagreeable rather than painful, and are described as queer, tightness or looseness of scalp, lightness or heaviness of the head, etc. Spinal tenderness and morbid fears common.—Neurasthenia.

(b) Headache appears and disappears in accord with emotional changes or in response to suggestion, often limited to a small spot at vertex (clavus). Various signs of hysteria may be present.—Hysteria.

ALTMAN.

HYSTERIA SIMULATING DISSEMINATED SCLEROSIS.

Charcot, Rendu, Souques and others have published so many cases of hysteria simulating disseminated sclerosis that further observation would seem without much interest, but the case related by Dr. Bonne (*Gazette Hebdomadaire*, Dec. 17, 1896) is interesting in that the neurosis while reproducing most of the symptoms of the organic disease was wanting in most of the cardinal symptoms of hysteria. The patient's first symptoms were like mild epileptic attacks, and began suddenly in May, 1891, apparently from a small emotional cause. They continued to return until three or four happened every day. A sort of aura of variable character heralded them constantly, or ordinarily a sense of constriction of the chest and a ball in the throat, sometimes a swelling of the abdomen. He came into the Hospital of Lyons in October, 1896, when he was still suffering occasionally from convulsive attacks, which were brought about under the influence of emotion or by pressure upon the hysterogenic zones. He was found to have difficulty of speech of a somewhat spasmodic character, but not typically scanning. This had begun two years previous to admission and progressively increased, though with no aphasia, and without motor trouble with the tongue. About the same time that this difficulty commenced he had also perceived a difficulty in walking. At the time of admission his gait was wavering and decidedly spastic; but this rapidly improved, as did his station, which was bad. There was a large tremor of the arms, disappearing in repose, controlled by small movements and made worse by large movements or ones demanding great precision of use

of the members. The hand-writing, while not typical, was also somewhat like that of a patient with sclerosis. All the tendon reflexes were greatly exaggerated; the plantar reflex sometimes was like a series of irregular jerks of the leg; there were both knee and ankle clonus easily developed, lasting some time and at once resisted by a voluntary effort of muscular contraction. There was no fibrillary twitch, and the decidedly hysterical symptoms were only a pharyngeal anæsthesia and sharply outlined and unvarying zones upon both fore-arms and on the right arm in which touch was not perceived. On the outside of the left calf there was an area of thermo-anæsthesia. Besides this, the visual field was contracted in both eyes and there was dyschromatopsia for all colors except red. There was an equal bilateral amblyopia and a permanent myosis with enfeeblement of reaction to light and distant vision. There was also slight nystagmus. The fundus was entirely normal. The author does not believe this a case of disseminated sclerosis with concomitant hysteria, but thinks it hysterio-epilepsy, in view of the history of the patient, the fact that difficulties of gait and speech not only were better from treatment, but presented certain points of decidedly hysterical character; again the occurrence of disturbance of color vision, the distribution of the sensory troubles, the persistence of the tremor in repose, all unite to favor this view.

J. K. MITCHELL.

HYSTERICAL CONTRACTURE OF THE MASSETER MUSCLE.

Verhoogen (*Jour. de Med. et de Chirug.-Prat.*, Oct. 25, 1896) reports a case of rare localization of hysterical spasm. The patient was a boy of 12 who had received from a comrade a slap on the right cheek. It was not very severe, but the following morning when the patient awoke he could not open his mouth. A month after the accident he presented the following symptoms: When asked to open his mouth he separated the lips, but the jaw remained immovable. Any attempt to separate the teeth immediately provoked severe pain in the right masseteric region. However, by patience and proceeding with great care and gentleness it was possible to slowly open the mouth to a reasonable extent, but during this procedure the masseters were observed to repeatedly contract. At the angle of the jaw there was an area of extreme tenderness, but if the finger were placed on the skin just beyond this, where touch was not painful, the skin could be pushed along and pressure made, as it were, beneath the tender place without causing pain. That is, the hyperesthesia was purely cutaneous. There were also areas on both arms in which the thermic sense was lost. The mother was a confirmed hysteric. Under chloroform the spasm relaxed and the joint was found to be absolutely normal. The contracture then was purely hysterical, the slight traumatism having acted simply as a determining and localizing agent.

It may be interesting to note that this case presents a striking antithesis to one reported by Charcot in one of his Tuesday lectures. In that case a woman, after giving her child a box on the ear, was stricken with hysterical paralysis of the offending hand with accompanying complete anesthesia. If one might imagine the two cases to occur simultaneously, we would have a dramatic incident, the moral of which, psychological, medical and ethical is self-evident.

PATRICK (Chicago).

A CASE OF HYSTERIA WITH ATAXIA CONFINED TO ONE LEG.

Charles W. Burr, M.D., narrates, in the *Medical News*, Nov. 14, 1896, the history of a man, aged fifty-six, who for about a year noticed "a peculiar sensation in the left leg as though worms were crawling over it." This lasted about two months and was followed by a feeling of extreme coldness in the entire left side. At times, when walking, he

loses feeling in the left foot and is unable to locate its position. Before entering the hospital while walking in the street, and without any preceding vertigo or headache or ill feeling of any kind, he suddenly fell unconscious as if shot. On examination in bed there is no evidence of either palsy or ataxia, but as soon as he gets upon his feet a certain motor difficulty is apparent. He has great trouble in standing, much increased by closing the eyes. The left knee and hips are alternately flexed and extended, and the trunk sways backward, forward, and laterally, but always to the left side. After a minute or two, if the eyes are shut, the movements of the left leg become quite wide in extent and he falls. In walking, which he can only do with the eyes open, he has great difficulty in putting the left foot where he wishes, the trouble being purely ataxic and not at all paralytic. He cannot follow a straight line, but always deviates toward the left. On the other hand he cannot turn around toward the left. So soon as he completely lifts the right foot from the ground and attempts to rotate the left upon the floor, the left leg begins to jerk, the body to sway, and he falls with some violence. He turns fairly well to the right. Under all conditions the ataxia of station is greater than that of motion. He can stand upon the right foot alone as well as, or even better, than upon both, and can stand upon the left only long enough to take a step with the right. There is no ataxia in the right leg, the disturbance of equilibrium occurring when he stands upon it being caused by the irregular movements in the left leg. All movements of the arms are executed well. In short, he stands and walks like a man drunk in one leg. He has, one may say, *astasia abasia* of one leg. There is anesthesia to touch, pressure, pain and temperature over the entire left side from crown to toe, and stopping precisely at the middle line. Taste sense is absent on the left half of the tongue and there is a partial deafness in the left ear. The visual field is contracted to a small area, corresponding to the macula lutea. Vision is $\frac{1}{10}$ for the right eye, $\frac{1}{15}$ in the left. On the third day after admission the hemianesthesia disappeared, but the contraction of the visual field of the left eye, the unilateral loss of taste and partial deafness and the motor trouble continued. The last symptom rapidly improved and on leaving the hospital ten days later he was able to stand quite well, though still unable to turn shortly to the left.

SHIVELY.

HYSTERIC NATURE OF COCCYGODYNIA.

Bremer (Med. Record, Aug. 1, 1896) enters a vigorous protest against the use of the knife in these cases. He considers the affection to be nearly always hysterical and cites two illustrative cases. Of one of these he says:

"On presenting herself to me for examination and consultation, this patient has the appearance of a healthy, well-preserved matron, whose looks do not betray the slightest trace of the Iliad of woes which she relates in a graphic manner. Knowing by experience with other cases that coccygodynia is almost always one of the symptoms of hysteria, sometimes apparently monosymptomatic, all the other manifestations of the disease being overshadowed or rendered dormant or insignificant by the overtowering dominance of one—the excruciating pain, I make the preliminary diagnosis of hysteria.

"Of course, I look for hysterical stigmata, but there are none; above all, there is an absence of anæsthesia of any kind anywhere, nor is there the slightest indication of a history pointing to hysterical attacks. This woman has been exceptionally healthy all her life. But on close examination I find that travelling and change of scenery lessen the pain, that at times she is slightly aphasic, that there is a tendency to a pulling back of the head, and that often she has "a lump in the throat." I add to this that she is of a gay temperament, and

that in spite of the overwhelming pain she has at all times, but which is particularly aggravating in the sitting posture, she does not present the aspect of a sufferer after having sat in my office for a number of hours, and I come to the conclusion that in the present case the coccygodynia is of an hysterical nature."

He says further:

"The text-books on medicine, and those on neurology in particular, describe the affection spoken of as being neuralgic or rheumatic in character. Probably there exists such cases, although among the dozen that during the last twenty-five years have come under my observation not a single one was of such nature. They were all symptomatic of hysteria, some of them apparently, but not in reality, monosymptomatic. For it is a striking fact that most women thus afflicted positively declare that there is nothing else the matter with them, though closer inquiry brings out the fact that a number of minor complaints exist or have existed, which, however, are ignored or have been forgotten. Hysteria is noted for the tendency to oblivion of ills that have passed.

"In all cases of coccygodynia that I have seen, a history could be elicited, if not of hysteria proper or some allied neurosis in the ascendants, at all events of the existence of the hysterical temperament. In all of them an immediate or provoking cause, a provoking agent (*agent provocateur* of Charcot-Guinon) could be demonstrated. A trauma, severe and prolonged emotional and intellectual strain, infectious diseases, convalescence, parturition and lactation, chronic intoxication (alcoholism, saturnism, etc.) can generally be shown to have existed before at the time of the cropping out of the trouble. The case briefly reported above is one of traumatic (monosymptomatic) hysteria. The several therapeutic procedures (insignificant in the healthy) acted like so many distinct shocks and provoking agents. The administration of the anæsthetic (in some predisposed individuals this alone suffices to bring about hysteria, transient or lasting) in conjunction with the wound, and later on the irritation set up by the introduction of the electrode into the rectum, sufficed to aggravate a condition which, if left to itself, would probably have remained within the bounds of toleration."

Regarding the evidence of hysteria the following statement may be of interest:

"What Charcot and his school have not mentioned in their classic delineations of the syndromes of hysteria are the spastic tendency of the retractors of the head and a trace more or less noticeable of aphasia. I consider them as stigmata of a subtle character, the anæsthesias being of a coarser kind. They are very common in hysterical females and sometimes the only obvious ones in a chaos of indistinct and undefinable malaise."

We think that the experience of all surgeons and neurologists would scarcely be in entire accord with that of the author, as presented in the following paragraph:

"I do not mean to say that never and under no circumstances has the removal of the coccyx been successful in curing pain. Perhaps there are cases in which the operation has been beneficial. Personally I do not know of any. Even in cases of success the question is legitimate: Would not other and simpler means have been equally effective? Generally speaking, the results of coccygodectomy are as hopeless as neurectomy in facial neuralgia."

PATRICK (Chicago).

ON SENILE EPILEPSY AND GRIESINGER'S SYMPTOM DUE TO BASILAR THROMBOSIS. By Prof. Naunyn. *Journal de Neurologie et d'Hypnologie*, Nos. 1 and 2.

Griesinger has pointed out that compression of the carotid arteri-

ies may cause epileptiform attacks if there is basilar thrombosis or analogous lesions of the *circulus arteriosus Willisii*. Cancato thinks that this phenomenon can be utilized for making the diagnosis of endarteritis of the cerebral arteries, as he could elicit it in ten individuals presenting arterio-sclerosis while this compression of the carotid arteries remained without effect in ten (evidently healthy) men between the ages of 23 and 25. Naunyn reports three cases in all of which he was able to call forth epileptic attacks by compression of the carotid arteries. Two cases and probably also the third concern senile individuals (65 to 70 years). One suffered from attacks of vertigo. The second one had typical epileptic seizures, which had come on at the age of about 53 years. In all three patients there was marked arterio-sclerosis. In case 3 the compression of the carotid arteries had evil effect (not given in detail since the report of this case is left out, evidently by some mistake), and the post-mortem examination in this case showed that all arteries of the base of the brain were healthy previously.

The observations made lead N. to the following conclusions:

1. The compression of the carotid arteries may prove dangerous and ought therefore not to be made use of for diagnostic purposes.

2. It is true that compression of the carotid arteries may call forth epileptiform attacks if there is thrombosis of the basilar arteries or some analogous lesion of the *circulus arteriosus Willisii*; but this compression can also give rise to similar paroxysms, when the arterial circulation of the brain is disturbed by a *general condition* such as valvular insufficiency, diminution of the energy of the heart and generalized arterio-sclerosis. Accordingly the phenomenon described (Griesinger's symptom) has no such definite diagnostic value with regard to the condition of the cerebral arteries as Cancato ascribes to it.

ONUF.

EPILEPTIC AURAS.

M. J. Durand (Thèse de Paris, 1896) makes a provisional classification of auras. He finds that an aura is present in 71 per cent. of epileptics, and is a little more frequent in woman than in man. It is constant, when it exists at all, in 92.9 per cent., invariable in 91.5 per cent., simple in 52 per cent. and complex in 48 per cent. The aura must be considered as having a central origin. Its localization will sometimes serve to indicate the site of the epileptogenic lesion. Attempts to control the occurrence of the fits by interrupting the course of the aura have given very uncertain results; however, the presence of a constant and invariable aura at least renders the prognosis of epilepsy less bad, and sometimes, if the warning be sufficiently long, will allow him to follow his ordinary course of life.

MITCHELL.

EPILEPTIC AMNESIA.

M. Seglas (Société de Méd. Legale; Séance, March 9, 1897) reports two observations upon retro-active amnesia in epilepsy, which are particularly interesting as both of the cases were pure epilepsy; alcohol, traumatism and other etiological factors could be entirely excluded.

The first patient was a man, 28 years of age, with bad family history, but personally well until 14 years of age, when he had his first epileptic attack. No return took place until he was 23 years of age, when the attacks occurred about once a month without being accompanied with any unusual symptoms. At one time at table he wounded himself with a knife during an attack. Upon recovery of consciousness, and afterward, he was unable to remember anything which had occurred during the morning of that day.

The second example was a patient at the Salpêtrière, 40 years of age, who had been the subject of epilepsy for five years. The attacks were mild so far as motor manifestations were concerned, but the con-

dition of semi-consciousness persisted for a quarter of an hour. Upon his recovery, and afterwards, he was unable to recall anything which had happened during the attack and for some hours preceding the paroxysm.

The author makes clear the distinction between these retrograde amnesias and the cases where the crisis is preceded by vertigo or delirium; and there should also be a distinction from the post epileptic confusion, which are so common in cases in which alcohol and traumatism play a part.

The medico-legal aspect of this state of forgetfulness is of obvious importance.

MITCHELL.

Gaz. Hebdom. de Méd. et de Chie., March 18, 1897:

THE TENDON REFLEXES IN THE POST EPILEPTIC STATE.

G. Pieraccini in studying the behavior of the tendon reflexes during and after epileptic convulsions, came to the following results: Immediately after a convulsive attack in the period of coma, the tendon reflexes are almost abolished, or at least decidedly diminished. After an interval of 5 minutes to half an hour they reappear, feeble at first, then slowly increase until the patient is completely over the attack when the reflexes are found slightly increased. The diminution of the reflexes is proportional to the duration and intensity of the attack. In even lateral convulsions the abolition of the reflexes is limited to the affected side. —Settimana Med. dello Sperimenale, No. 32, 1896.

W. C. K.

CONSTANT SIGNS OF EPILEPSY.

MM. Mairé and Vires read before the Académie de Médecine (Medical Week, Feb. 5, 1897), a paper on epilepsy, in which the authors say it has been their endeavor to find constant signs of that disease, whose existence would permit of the certain diagnosis of the epileptic nature of an attack, whether of convulsive or larval epilepsy. This is especially important since epilepsy of the convulsive form may be simulated so well as to make it difficult for the most careful examination to detect the imposition. The authors from study of a large number of cases have reached the following conclusions:

Even a single attack of epilepsy is associated with an increase in the nitrogen and phosphates excreted.

The course of the general temperature is characteristic, being lower than normal during the convulsive period and during or following sleep, with an increase upon awakening and a gradual rise to the normal. The urine is hypotonic after the attack.

During the periods between the paroxysms the urine is hypotonic also. The fatal dose of the urine of a healthy person varies between 45 and 80 cubic centimetres, whereas that of the epileptic is between 150 and 250 cubic centimetres.

The urine of larval epilepsy is constantly hypotonic, the fatal dose ranging between 150 and 480 cubic centimetres.

This constant hypotonicity in epilepsy is therefore an important, permanent sign from a medico-legal point of view, especially as it may be utilized for a diagnosis of the obscure larval form of epilepsy.

J. K. MITCHELL.

PSYCHIATRY.

ESSENTIAL ACROPHOBIA.

Acrophobia (fear of high places) in a degree scarcely to be designated as pathological, is very frequent in nervous and timid people. As a marked or prominent symptom in the more pronounced cases of neurasthenia it is far from rare, but entirely dissociated from other nervous manifestations it is very uncommon.

Gelieanu (Jour. de Méd. de Paris, Oct. 4, 1896) reports such a case

in the person of a young Russian. If the dates given in the paper are correct, the trouble began when the patient was only 10 years old, and without apparent cause. There was a neuropathic family history, but the first attack occurred on a cliff where the boy had been accustomed to go daily. While regarding the declivity, he was suddenly taken with vertigo, weakness and an agonizing oppression. With difficulty he regained the dwelling, which was near at hand, and was obliged to go to bed. The next day he could laugh at his fears and had no more attacks for a year. Then he was again seized with the unaccountable fainting terror and for the remainder of the summer was unable to walk on any elevation, even a hill. Seated in a carriage he had no trouble. Following this time he lived for six years in a flat country and was perfectly well. He then had occasion to return to the scene of his first attack when the trouble immediately returned, and was more severe than ever. He was now as bad in a carriage as on foot, and even to look out of the window of a house situated on a bluff brought on the insupportable agony with a feeling of impending death. For two years after this he was the subject of this abnormal fear. Looking at a photograph or painting of mountains would bring on an attack. He began, too, to be nervous and apprehensive in a crowd and felt comfortable only at home or on a plain. A combination of tonics and sedatives greatly improved his condition, but there is no assurance against a relapse.

PATRICK (Chicago).

A STUDY OF THE BLOOD IN GENERAL PARALYSIS. By J. A. Capps. American Journal of the Medical Sciences, June 18, 1896.

The conclusions based on the observation of 19 cases are:

1. The hæmoglobin and red corpuscles are always diminished.
2. The specific gravity falls slightly below the normal.
3. Most cases show a slight leucocytosis, amounting on an average to about 22 per cent. above the normal. Early cases may have no leucocytosis whatever.

4. In the differential count a decrease is found in the lymphocytes along with a marked increase in the large mononuclear cells. The eosinophiles in a few cases are very numerous.

In convulsions and apoplectic attacks (1) the red corpuscles and hæmoglobin are usually increased at the time of a convulsion. During an apoplectic attack of long duration they are both somewhat diminished.

2. The specific gravity is variable, sometimes increasing, sometimes diminishing, at the time of an attack.

3. There is a leucocytosis after convulsions and apoplectic attacks, which is as sudden as it is usually pronounced. It certainly does not appear until within a very short time preceding the convulsion, probably not before it actually takes place.

4. The degree of leucocytosis and the period of its continuousness, as a rule, vary directly with the length and severity of the attack.

5. In the production of the leucocytosis the large mononuclear cells are increased relatively more than any other variety.

6. The fact that after convulsions and apoplectic attacks in general paralysis there is not only an increase in the number of white cells, but a change in their character, as shown by the differential count, and at times abnormal cells appear, is an argument against the theory that leucocytosis is merely a change in the distribution of the white corpuscles.

SHIVELY.

DISTURBANCE OF SENSIBILITY IN MELANCHOLIA.

Dr. Pierre Dheur finds (Thèse de Paris, 1896, No. 62) that disturbances of sensation are the rule in the course of melancholia, whether the form of the disease be the depressed, agitated or dull type.

There is one disturbance of sensation which is constant, viz., the loss or considerable diminution over a greater or less surface of tactile sensibility. The analgesia is often so extensive as to leave but a small portion of the surface healthy. The one area which is seldom affected Dr. Dheur finds to be the upper-lip. The analgesia is sometimes localized in a single limb, often on only one aspect of a limb; the lesions are frequently, but not always, symmetrical. Lessening of thermal sense is noted chiefly in agitated melancholia. The reflexes are also sometimes lost in these patients, and four times Dr. D. has seen the patellar reflex exaggerated. In one-third of the cases hyperæsthesia is present, usually of small extent and very variable in situation. Olfactory, gustatory and auditory sensibility are all occasionally found affected. Diminution of vision often coinciding with narrowing of the visual fields was also observed. Respiratory movements were noticed to be irregular in some cases, and often less frequent than in the healthy.

These interesting observations, like so many of the disturbances of sensibility in hysteria will probably not be found to be so usual in this country as in France.

J. K. MITCHELL.

MENTAL CHANGES IN GRAVES' DISEASE. By A. Mande, L.R.C.P. Jour. of Ment. Science, January, 1896.

Dr. Mande reviews twenty cases of exophthalmic goitre, studying the mental phenomena. He considers only one of the twenty as definitely insane, yet regards only two of the twenty as having normal mental state. He describes as common an irritable restless state that he calls neurasthenic, with chorea of ideas and loss of ability for connected thought. Personally, we are impressed with this description in its alliance to the irritable prodromal state of senile dementia, choreic dementia and paretic dementia.

THE DISORDERS OF THE MUSCULAR SYSTEM IN INSANITY. Theodore H. Kellogg, M.D. Medical Record, Aug. 15, 1896.

In a timely paper on the above subject, Kellogg points out the various disorders manifested in the muscular apparatus of the insane. According to their clinical frequency we meet with, 1st, atrophy; 2d, tremors; 3d, contractures; 4th, spasms; 5th, cramps; 6th, pareses and paralyses. These categories apply to the voluntary muscular system. Atrophy is very much more frequent in insanity than is commonly supposed, and sometimes reaches excessive grades. It often takes place very rapidly, as in acute delirium. In the various toxic insanities atrophy is commonly the result of trophic lesions in the central nervous system. In the diathetic insanities gout, rheumatism, syphilis, tuberculosis, etcetera—muscular wasting is not infrequent. In general paresis and alcoholic dementia, polyneuritis often induces atrophy. In idiocy, imbecility and cretinism this symptom is common. Simple atrophy may, of course, also arise from disuse as in bed-ridden cases.

Tremors of various kinds, coarse and fine, partial and general, constant and interrupted, or "intentional," are very frequent among the insane. During repose they usually disappear.

Contractures of varying degree are common in insanity. They may be the result of habit, such as fixed postures, for instance in melancholia, or be due to central nervous lesions, as in organic dementia, syphilitic and alcoholic insanity and other forms, or be caused by joint troubles, as in rheumatic insanity.

Spasms, both tonic and chronic, are frequently encountered, but the latter are not as common as the former. Often they resemble, or are identical with, the psychic and convulsive tics. Tonic spasm of the orbicularis palpebrarum, and of the sterno-cleido-mastoid and trapezius is common. Esophageal and pharyngeal spasms also occur. The

spasmodic clonic movements of choreic insanity are to be classed under this head.

Strabismus is common in all types of insanity and the permanent divergent squint Kellogg holds to be of unfavorable diagnostic import.

Cramps of all kinds are among the muscular anomalies to be noted among the insane. The gastrocnemius is, perhaps, most frequently affected. These cramps are often painful and the source of insomnia, especially in neurasthenic and alcoholic cases. Tetanoid, epileptoid and cataleptoid may, among others, be distinguished.

The *pareses* and *paralyses* are probably the most serious of the muscular disorders. They are of every variety and form, according to the location of the lesion in the central nervous organs and peripheral nerves, and are of the same general character as those resulting from nerve lesion without mental disease. Therefore, it is unnecessary to review them more fully here. The well-known automatic movements so commonly seen in various forms of insanity, imbecility and idiocy, Kellogg also classes under the muscular affections, but many neurologists and alienists will doubtless not concur in this view.

The sensory disturbances of the muscular system are also concisely spoken of. Among these are to be mentioned the deviations in the muscular or kinæsthetic sense, namely, the muscular anæsthesias and hyperæsthesias, myalgias and lumbago. To the anomalies of the involuntary muscles, only a few lines are devoted.

Altogether Kellogg's paper is important and timely, in as much as the physical signs of demented persons are often neglected or overlooked, though they usually form factors of weight, both in the treatment and prognosis of nearly all cases which exhibit them.

STERNE.

ATTENTION AND "AUTOMATISMS" (IRRELEVANT MOTIONS).

Lindley (Clark University) describes a series of experiments with school children and adolescent pupils in the kindergarten and primary grades of the Boston Normal Training School. They were made in conjunction with President Stanley Hall for the purpose of finding the frequency of automatisms during various forms and degrees of attention. In all there were 662 cases tested (some subjects counted more than once), 235 were children of twelve years of age or under, and the rest were nearly all under twenty years of age. 421 were females, 241 males, and of the 235 children there were 117 girls and 118 boys. Of adolescents 304 were females and 125 males.

The first form of experiment was the drawing of straight and zig-zag lines between perpendicular parallel lines extending between two horizontal parallel lines. Work continued fifteen minutes followed by an interval of five minutes, and then resumed for ten minutes more. They made large and small lines alternating for about equal lengths of time. The children were in groups of six or eight in number.

The average number of "automatisms" (irrelevant acts) per hundred was found to be, for children 124, adolescents 110. About half of the total number of automatisms belonged to parts of the head and face. Of single groups recorded, fingers and feet led in both children and adolescents. The lips ranked high in both lists. The head "automatisms" were much more frequent in children than adolescents, and the mouth, lips and tongue were not far behind. Swaying, the commonest of the body "automatisms," was almost entirely restricted to children. Playing and drumming with fingers were not so frequent with children as with adolescents. Girls led in swaying and finger automatisms, but boys led in tongue, feet and hand movements. The average number of "automatisms" per 100 girls was 179, boys 181.

Some groups gave the "automatisms" of writing, recitation, pub-

lic recitation, reading, conversation, attention, study, difficult recollection and greatest effort. In these the following points were observed: Automatism varied somewhat with the nature of the activity in question, *e. g.*, in writing, lips and tongue furnished 46% of all the automatisms; in reading, body, head, hands and fingers comprised more than half of the whole number; in recitation, feet, fingers and body led; in study it was fingers, eyes, hands and jaws, and so on. There were few automatisms of those muscles engaged in a given task. But when those parts which show greatest frequency of automatisms were pressed into actual performance of work, as the fingers in writing and the vocal organs in reading, the number of automatisms were not thereby apparently diminished. The average number of automatisms per 100 persons appears to increase slightly with the intensity of effort, *e. g.*, recitation, public recitation and conversation show an average of 120 automatisms per hundred; but attention, study, difficult recollection and greatest effort yielded 136. Also automatisms increase in number and intensity with age in the kindergarten—the class containing those of ages $3\frac{1}{2}$ to 4 showing fewest and feeblest. In the primary group (ages 6 to 7) there was a marked falling off in automatisms as compared to the older children in the kindergarten. The automatisms of the youngest were chiefly of postures. Also the automatisms were more pronounced in making small movements, and in nearly every case the automatisms showed a somewhat rapid increase toward the end of the work period. The automatisms of the head (neck muscles), body, legs and arms were rather more than twice the number in children as compared to adolescents, being 9.7% and 4.4% respectively of all automatisms.

CHRISTISON.

From American Journal of Psychology, July, 1896.

THERAPEUTICS.

REMOVAL OF THE TRUNK OF THE SUPERIOR MAXILLARY NERVE AND MECKEL'S GANGLION. McKay. Australasian Medical Gazette, January, 1897.

The patient, a man of 41, had 22 years before been shot in the left side of the face, the ball never having been removed. Three years before he came to McK. he had begun to suffer from severe neuralgia in the left side of the face. The cicatrix had first been dissected out, and the ball taken away, and later the Gasserian ganglion had been removed, without giving permanent relief. MacK. cut down and secured the infraorbital nerve, and with it as a guide, chiseled through both anterior and posterior walls of the antrum, removed the floor of the infraorbital canal, dissected out and took away a section of the nerve with Meckel's ganglion. The presence of the ganglion was shown by microscopical examination. Owing to bad surroundings, the patient got erysipelas in his wound, but recovered, and was free from neuralgia for three months. Later he had another attack of erysipelas, and has suffered since from neuralgia. It is considered by the author as remarkable that the pain persisted after the removal of the Gasserian ganglion in the earlier operation. He seems to think, however, that there is some question as to whether it was really removed.

C. L. ALLEN.

NERVE SUTURE. Gaz. Hebdomadaire de Med. et de Chi., February 7, 1897.

In a thesis Ehrmann reviews the history of suture of widely spread nerve ends, which he considers necessary when the distance between the two extremities is greater than 2 c. m., believing that some sort of a way must be prepared from the centre to the peripheral end. The proceeding is only necessary when there has been a loss of nerve sub-

stance so considerable that the two ends cannot be brought into exact opposition.

Several proceedings have been suggested to obtain this result.

Letivant suggested the attachment of the lower end of the nerve to the free extremity as an intact neighboring nerve—a nervous graft.

Vulpian, and others, made experiments on nerves by transplanting a portion of a nerve of an animal between the two widely spread ends, but for distant suturing this has not been found successful.

Van Lair and Assoky furnishes a sort of bridge to the regenerated by attachments of silk or cat-gut threads along which the fibres of the regenerated nerve might follow. These authors succeeded in thus bridging a gap as long as $3\frac{1}{2}$ c. m., and they believe that the plan is a particularly feasible one. The threads persist (even when made of an absorbable material like cat gut) for a sufficient length of time to allow of the growth of approximal ends of the nerve along their course.

MITCHELL.

NERVE SUTURE AND OTHER OPERATIONS FOR INJURY TO NERVES OF THE UPPER EXTREMITY. A. J. Ochsner, M. D. *International Jour. of Surgery*, December, 1896.

Every severed nerve should be sutured, even after years, and the earlier the operation is performed the better. If neither sensation nor motion is established within a year the nerve should be again exposed, the cicatricial tissue removed and the ends again sutured. The ends should be clean cut and contain neither crushed tissue nor cicatricial tissue. Tension must be avoided. The wound must heal without suppuration to secure the best result. Hemorrhage should be controlled perfectly to prevent intervening clot. Carefully prepared cat-gut is the best material. After suturing the ends, either direct or "a distance," it is well to stitch a fold of fascia over the united nerve-ends. The extremity should be placed at rest. The external incision should be ample. Castor oil taken in large, daily doses often relieves pain due to traumatic injuries of nerves.

FREEMAN.

A CASE IN WHICH THE SPINAL CORD OF A RABBIT WAS SUCCESSFULLY USED AS A GRAFT IN THE MEDIAN NERVE OF A MAN. By Mayo Robson. *British Medical Journal*, October 31, 1896.

The patient fell on a scythe and cut his upper arm at the inner and lower part. All the muscles acting on the wrist and hand, which had their nerve supply from the median and ulnar nerves, were paralyzed, while those supplied by the musculo-spiral were capable of acting. There was also marked alteration of sensation. The extensors reacted to faradism; the flexors showed no reaction. It was evident that there had been a complete division of the median, ulnar and internal cutaneous nerves. About seven months after the accident incision was made along the line of the cicatrix, and the two ends of the internal cutaneous nerve were sutured; those of the ulnar were united by grafting strands of the sciatic nerve of a rabbit so as to fill up the gap. The ends of the median could not be brought nearer one another than two and a half inches, and a portion of the spinal cord of a rabbit just killed was used as a graft to connect them. The operation was performed January 30, 1890; on February 10 the patient could feel the scratch of a pin on the flexor aspect of the first phalanx of the thumb, and at the root of the index finger. On February 17 sensation had returned over the whole of the palmar surface of the thumb, and the proximal phalanx of the index finger. When the patient was seen six years after the operation the muscles of the affected limb were found to have nearly their normal volume. The only weak muscle was the abductor of the thumb. Sensation was perfect. SPILLER.

TRAUMATIC PARALYSIS CURED BY STRETCHING THE NERVE.

Dr. Mouchet (Medical Week, June 19, 1896) reported briefly to the Paris Academy of Medicine the case of an alcoholic who had paralysis of the radial nerve, following a blow. The paralyzed nerve was stretched two months after the accident, the operation being followed on the first day by perfect return of the sensation where it had been completely abolished, with complete recovery in ten days.

At the Academy of Medicine, July 28 (Medical Week, July 31, 1896) Dr. Perier reported on three cases of Dr. Mouchet's, two of paralysis in the facial and one in the posterior tibial nerve. Dr. Mouchet had stretched the nerves with good results. In the first two there was no compression, in the third the nerve was compressed, and the relief was probably as much due to the removal of this pressure by dissecting out the nerve from the tissues about it, as to the stretching, and it is possible that there may have been some influence of the same kind in the first case, and that this may have had quite as much to do with the relief as the moderate degree of stretching to which the nerves were subjected. MITCHELL.

COMPRESSION OF THE CAROTIDS IN EPILEPSY.

Compression of the carotids was introduced early in the present century as a remedy for epilepsy, but has never attained much vogue. Before the time of Galen it was known that it produced stupor and collapse resembling apoplexy; and if carried further caused convulsions. Kussmaul and Tenner, in their classical monograph, regarded it as of little use. Because ligature of both common carotids, or a large and sudden loss of blood causes in rabbits epileptiform convulsions, and because the profuse bleedings formerly employed gave rise to spasms, they stigmatized compression of the carotids.

But it seems to me that the cases are very different. In ligature the blood is permanently or for a long time cut off from the parts of the brain supplied by the arteries—that is, until the collateral circulation is established by means of the vertebrals; whereas, in compression the arrest of the blood is only temporary or partial.

Now, whatever theory we may adopt as to the causation of the epileptic seizure, there is little doubt that during the period of unconsciousness there is a very decided hyperæmia of the brain, either localized or general; and it is also true that in many severe seizures, as in many cases of status epilepticus, there is a marked, forcible and increased pulsation in the carotids. In my own experience several times the tension was so great that compression was extremely difficult. It is in such cases that I have derived the greatest benefit from compression of the carotids. For convenience of description an epileptic seizure may be divided into three stages: (1st) precursory, including incubation, aura, etc.; (2nd) tonic stage, including tonic spasms, unconsciousness, partial or complete, etc.; (3rd) clonic stage, clonic convulsions, laryngismus, trachelismus, fixity of chest walls, distension of veins of neck, redness or cyanosis of face, dilatation of pupils with complete irresponsiveness, and entire abolition of consciousness. In this third stage the pulsation (increased tension) is most marked, and it is in this stage only that I have tried compression, in accordance with the usual custom. By it we not only diminish and regulate (control) the amount of blood going to the cerebrum, but at the same time increase the flow of blood in the vertebral arteries, thereby relieving anæmia of the medulla (?) causing the respiration to become easier, and promoting the oxygenation of the blood, with immediate benefit to the nutrition of the brain.

Reynolds, J. Lewis Smith and others think that these results are due not so much to compression of the carotids as to pressure on the sympathetic nerve, which causes contraction of the cerebral vessels.

By pressing the thumbs backward and a little inward we can avoid the nerve, which passes down by the side of the vertebral column. In this way you also avoid pressure on the pneumogastrics, which lie behind and a little external to the carotids. Care must also be taken to avoid undue pressure on the trachea.

Flint recommends pressure on one carotid only, at first, on the side where the convulsions first begin, or are most marked. If that proves ineffectual then apply pressure to both. In conclusion pressure on the carotids seems of some use, first, in very severe or prolonged seizures, and second, in status epilepticus, where it may sometimes abort the attack, and in most cases shortens the duration or diminishes the number and severity of the fits.

The bibliography of the subject is meagre.

J. M. KENISTON, M.D. (Middletown, Conn.)

ANTITOXIN TREATMENT OF TETANUS.

Ridge (British Medical Journal, September 12, 1896) reports a case of a lad of 16 who recovered under injections of tetanus antitoxin, out as the period of intubation was 13 days, the chances of recovery were fairly good under any mode of treatment. No marked effect from the injections could be noted, but after the fourth or fifth dose he slowly and steadily improved. PATRICK (Chicago).

CALCIUM CHLORIDE FOR ITCHING.

Savill (British Medical Journal, September 19, 1896) reports remarkable success in the treatment of itching from the administration of chloride of calcium in doses of 20 to 40 grains three times a day. The ordinary dose of the drug is 15 to 20 grains, but the author has found larger amounts much more efficacious. He says it is well borne if given after meals in a wine glass of water. To cover the salt taste he administers it in 1 dram of tincture of orange peel and 1 ounce of chloroform water. The diet should, at the same time, be regulated, no beer, sugar or sweets, and but little meat being allowed. When recovery has been accomplished the remedy should be gradually, not suddenly, withdrawn. The author is not yet able to tell precisely which cases of pruritis are most amenable to this treatment, but it succeeds in many and he advises its trial in all. PATRICK (Chicago).

DUBOISINE SULPHATE IN THE TREATMENT OF PARALYSIS AGITANS. By Prof. X. Fraucotte. *Journal de Neurologie et d'Hypnologie*, No. 5.

F. bases his results upon observations in four cases. He concludes that Duboisine possesses an actual (*réelle*) influence against the tremor of paralysis agitaus. Unfortunately this influence which manifested itself in all four cases, is rather ephemeral; two or three days after the suspension of the drug, sometimes even earlier, the tremor reappears with about the same intensity as before.

Duboisine diminishes also, but to less extent and less constantly, the rigidity and the distressing symptoms accompanying it. In view of the inefficacy of all other treatment, the administration of the drug is to be recommended in this disease, since it at least brings some relief and revives the hope of these patients. F. recommends to give the Duboisine Sulphate in doses of $\frac{1}{2}$ milligrammes from 3 to 6 times daily. He says, that although he has the impression that from 2 to 3 milligrammes *pro die* are required to produce a marked effect, he would not deny that smaller doses might be sufficient. In one case the daily dose was twice brought up to 4 milligrammes, when dryness of the throat, visual disturbances and vertigo resulted, which symptoms soon disappeared, however, after discontinuance of the administration or after lessening of the dose. ONUF.

OBSERVATIONS ON THE USE OF APOLYSIN. D. G. Greif. New England Medical Monthly, November, 1896.

Apolysin is well borne even in large doses and for long periods of time. Patients take it willingly. It does not irritate the intestinal tract even in large, continuous amounts. The urine remains normal during its exhibition. It causes no other disturbances. In an obstinate and protracted case of neuralgia of the fifth nerve it secured freedom from attacks of pain for seven months. The large doses that are required may deter some from its use. It is, however, just the length of time that the apolysin can be used and the large daily doses, up to 6 grains, and more, in which it may be employed, without the supervision of any toxic symptoms, that are its great advantages over other antineuralgic remedies. In hemicrania spastica and hemicrania angio-paralytica also, it has done most excellent service—one grain in dry powder being given as soon as prodromal symptoms appear. In many the threatened hemicrania was cut short. If it nevertheless appeared, a second grain an hour later and a third two after that, if necessary, almost always produced the desired effect. In 64 cases of hemicrania it was only necessary to use 3 grains in 8. When the apolysin was given during the aura 1 or 2 grains was almost without exception sufficient. When given in the evening patients sleep very soundly and think they have taken morphine—thus it appears to have a certain narcotic effect. The intervals between the attacks of hemicrania increased in length under the apolysin treatment and cases that the first time required three grains did well with two or even one in subsequent attacks.

FREEMAN.

THE SERUM THERAPY IN NERVOUS AND MENTAL DISEASES. Mairet and Vires. Nouveau Montpellier Medical. No. 8, 1897, p. 14.

The authors conclude from a series of observations of the action of various serums upon mental and nervous diseases the following:

1. Pure artificial serum by hypodermic injection has no appreciable therapeutic effect in epilepsy nor in mental diseases.
2. Artificial serums with medicinal substances in solution by the hypodermic method are inoffensive and serve only to carry certain drugs into the general circulation more rapidly. Since drugs as potass. bromide, ergotine and phosphoric acids seemed to give no effects whatever. Urethane alone would seem to be of service. It controlled maniacal excitement more rapidly when given by the hypodermic method with serum than when administered by way of the mouth.
3. The blood serums of the rabbit and the dog gave some positive results, that of the latter being more potent.
4. Pure blood serum produces its chief physiological factor, local or general erythema in all respects similar to the described erythemas of serum therapy.
5. While serum therapy would appear to be useless in hysteria, epilepsy and in those forms of mental disease associated with exaltation, it is useful in cases where depression is the found. In such cases temporary stimulation is obtained which may be made to continue under repeated injections, but while amelioration is evident permanent cures were not obtained. The bodily functions were stimulated, which might account for the relief of the mental conditions.
6. The results are hopeful and are sufficiently positive to cause the authors to believe that in certain cases the extension of the methods might give more favorable results.

JELLIFFE.

SIMPLE MELANCHOLIA AND ITS TREATMENT. By H. E. Allison, M.D. Med. Record, Jan. 7, 1897.

In the above article, Allison considers melancholia and neurasthenia as closely allied affections, in which egoism or the habit of

self-introspection is a characteristic feature. He insists upon radical treatment of the usual bowel complications. Proper elimination of the intestinal contents reduces or prevents further auto-intoxication. At the same time internal antiseptics should be employed. Nourishing diet and sleep must be maintained. With the views expressed Ref. is entirely in accord. Notably the feature of auto-intoxication should be kept in mind. This is nearly always present and demands careful attention.

STERNE.

PATHOGENY AND THE TREATMENT OF PROFESSIONAL SPASM.

M. Vigouroux presented to the Académie de Médecine (La Médecine Moderne, Nov. 4, 1896) his views upon this neurosis, which he believed to be always caused by changes in the nerves and muscles, the pathologic sequence being as follows: a general disorder, dyscrasic, infectious or toxic, determining first an exaggeration of reflex excitability in muscles, nerves and sensorial tissues. The lesions, in the next place, produce functional disorder directly by diminishing the blood supply, and secondly the sensibility of power of the muscles, by provoking a reflex spasm. He makes a general division of these functional palsies according to the seat of the peripheral lesion in each, and considers that the lithemic tendency is the most frequent general cause.

MITCHELL.

PARALDEHYDE. (Squibbs Ephemeris, Jan., 1897.)

It is a wonderfully innocuous drug, and where large doses are needed it can be well tolerated. It is an exceedingly efficient calmate and hypnotic. To illustrate these points the following cases may be cited: Case I.—A female, aged 81, melancholic, with suicidal tendency was treated with paraldehyde after numerous other sedatives had been tried with even injurious effects. This patient for several months took more than 1 ounce (once 4 ounces) in the twenty-four hours. The medicament not only gave great relief but proved a most suitable exhibit, for the patient recovered and remained well for three years. Case II., a female, aged 19, had been epileptic from childhood. The fits had lately become more frequent, often occurring two or three times a week. One peculiarity about the case was the prolonged aura. When the fit was to happen she felt upon awaking in the morning very ill, but could not describe any special sensations. Towards mid-day or in the afternoon the general convulsions seized her. The question of warding off the fits forced itself upon attention, seeing that so long an interval of warning was given. The idea of producing sleep then suggested itself, but how to avoid the danger of sedatives presented itself as a most serious difficulty. The favorable experiences of paraldehyde made one hope the obstacle could be avoided, and the result was most surprising. After the first dose of 15 minims she was sound asleep in five minutes and awoke refreshed with all disturbance gone. She has still the threatenings, but they are not so frequent, and has on several occasions had intervals of one month. For more than a year she has had no fit, except once when no paraldehyde was obtainable. She never requires over thirty minims (Dr. W. D. Aitken). A disagreeable feature of the drug is that it scents the breath strongly for about twenty-four hours. A point in dispensing is that the addition of a few drops of alcohol renders it perfectly miscible with water; any flavoring tincture can be used for this purpose (Dr. W. Mackie).

FREEMAN.

THE
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Nervous and Mental Disease

AMERICAN NEUROLOGICAL ASSOCIATION.

Twenty-third Annual Meeting, held at St. John's Parish
Hall, Washington, D.C., May 4th, 5th, and 6th, 1897.

The President, Dr. M. A. Starr, in the chair.

Session of May 4th.

President's Address,

THE TRANSMISSION OF SENSATIONS
THROUGH THE SPINAL CORD.

By PROF. M. ALLEN STARR, M.D., Ph.D.

Gentlemen:—

In taking the Chair to preside over this meeting, I feel that I must first express to you my thanks for the honor you have done me in choosing me as your chairman, an honor which I fully appreciate as I read over the list of names of my predecessors in office—men who have made American neurology respected in every land. I trust that you will aid me in this meeting in maintaining the high standard of our discussions, and thus secure the success of the twenty-third annual meeting of this society.

The subject which I wish to present to you this morning is the transmission of sensory impulses through the spinal cord, a subject which is still extremely obscure and by no means as yet accurately determined. I think that the conclusions derived from physiological experiments upon the lower animals are open to grave criticism, first,

because of the fact that the majority of such experiments performed years ago were done without antiseptic precautions, and were attended by secondary septic inflammations of the cord and its meninges, which invalidated the conclusions drawn from them; and secondly, because of the great variation in the anatomical structure of the cords of the lower animals and of man. It seems, therefore, necessary to rely almost entirely upon pathology for the data in determining the facts.

While recent investigations have abundantly confirmed the general results long known as ascending degeneration in the spinal cord after lesions of the nerve roots and after lesions of a transverse nature, the recent methods of investigation, especially the staining methods of Weigert, Van Gieson, and of Marchi, have furnished some interesting results. Everyone who has studied the degeneration process in the cord has been aware of the fact that in a degenerated tract there are always found many healthy nerve fibres, but it was due to Marie that this mixed character of the various columns of the cord was first definitely brought to our notice, and his results have been abundantly confirmed by the investigations with the aid of the Marchi method. It is no longer possible to speak of the columns of the spinal cord as being exclusively ascending or descending in the direction of their fibres. We know now that all columns of the cord contain fibres of greater or less extent passing both upward and downward, and that mingled among these fibres there are very many others whose function is purely associative between the various segments. Even in the long pyramidal and anterior median descending motor columns of the cord there are very numerous ascending fibres, and in the posterior columns, long supposed to be ascending only, we now distinguish the descending comma-shaped columns of Schultze, the descending posterior marginal columns of Flechsig, and the association fibres entering the limiting layer along the gray matter of the posterior commissure. Recent investigations by Hoche have shown the existence of many asso-

ciation fibres of limited extent in the direct cerebellar columns and in the columns of Gowers, and, therefore, it seems evident that every column of the cord contains fibres which are passing in both directions.

If we study carefully the result of degenerative processes following transverse lesions, it becomes evident that the degeneration, though very intense near the lesion, becomes very much less so as we get further from the lesion, no matter at what level the latter may lie. It is thus proven that the number of short tracts in the cord is far greater than the number of long tracts, and inasmuch as these short tracts can only be nerve roots, or else tracts of association, it is evident that there are but few very long fibres extending through the cord to the brain.

When the long tracts are carefully studied by the process of Marchi, it again becomes evident that the number of really long continuous fibres is relatively small. The long motor tracts are well established, but the sensory tracts are not so clearly defined. It has been the fault of many observers, who have studied ascending degenerations, to have described these degenerations only as high as the fourth or third cervical segment. This is probably owing to the fact that at autopsies the first, second and third cervical segments of the cord are commonly divided obliquely in getting out the brain, but in the cases in which this error has been avoided, and sections have been made of the second and first cervical segments, it is quite remarkable to discover how very much the degeneration in the so-called long ascending columns, which is very extensive in the fifth and fourth cervical segments, is reduced in size at the first cervical segment, a fact which proves that many of these long fibres supposed to reach the brain really terminate in the spinal cord itself.

Furthermore, when the oblongatal and pontine prolongations of these ascending tracts are investigated, it has been found that none of them are actually continuous with the fibres of the crura cerebri. In other words that there is no tract passing directly from the cord to the brain

cortex. Thus, the ascending fibres of the posterior columns all terminate in the nuclei graciles or cuneati, whence new fibres pass onward, either to the cerebellum or to the interolivary tracts. The fibres of the direct cerebellar column, which are considerably reduced in number before entering the restiform body, pass directly to the cerebellum. Edinger and Hoche have shown recently that the fibres of the ascending anterolateral tract or column of Gowers are also considerably reduced in number in the oblongata, and pass exclusively to the cerebellum through the superior peduncle, after traversing the formatio reticularis of the pons. While many fibres that lie in the anterolateral columns of the cord enter the formatio reticularis of the oblongata, none are known to pass continuously through the formatio reticularis, but all are supposed to terminate at various levels within the oblongata, pons, or tegmentum of the crura.

It must, therefore, be admitted that we do not know of any continuous sensory tract from the spinal cord to the brain, and even if the lemniscus is continuous from the oblongata to the cortex (and it must be remembered that some authorities deny this fact, and state that all the fibres of the lemniscus terminate in the corpora quadrigemina, or in the optic thalamus) it cannot be stated that the lemniscus forms a direct sensory tract from the cord to the brain, since it begins in the nuclei graciles and cuneati of the oblongata. These facts then compel us to believe that sensory impressions are conveyed through the central nervous system by means of short tracts, interrupted in their course, and not by long continuous fibres.

If we approach the subject from another standpoint, the same conclusion is forced upon us. The effect of an ordinary sensation in any part of the body, especially if that sensation be of sufficient importance to give rise to pain, is not merely a conscious perception. The primary effect is a multitude of reflex actions, entirely below the sphere of consciousness. Thus an ordinary painful impression will result in the retraction of the limb that is irri-

tated; in the setting up of vasomotor and trophic reflex actions, or, if the irritation is kept up, in a general sense, of uneasiness throughout the body, and even complex reflex actions to remove the source of pain; not to mention the quickening of respiration, the facial expression of pain, and automatic exclamations. All of these may be produced in an animal whose cortex is removed, or in a man when asleep or under the influence of ether. It is, therefore, evident that a sensory impression, if of sufficient severity, or if continuous, may practically throw into activity almost the entire subcortical nervous system. In other words, that a single sensory impression may be primarily distributed to all the motor centres through the entire length of the spinal cord and of the cerebral axis, in addition to its awakening cortical activity. This effect could not be attained were the sensory tracts from anyone region of the body continuous tracts to the cortex. It could only be attained by a mechanism which would provide for the termination of the single sensory tract at all levels of the cord, or by mechanism which would provide for the dissemination of a single sensation by means of short association tracts throughout all these levels, and as we have already seen from the study of anatomy and pathology, it is the latter provision which seems probable.

There is one other possibility of sensory transmission which need detain us but a moment. It is known that in the posterior horns of the cord a peculiar substance, the substantia gelatinosa, is present, which is also found at the termination of the descending branch of the fifth nerve in its entire extent, and hence has been thought to have a necessary connection with the reception of sensations. Golgi's researches have shown that this tissue consists of a very fine network of nerves and fibres, consisting probably of the terminal brushes of the nerves of sensation. This substance, therefore, has an important function in the reception of sensation. It may be stated, however, that it cannot have any function in the transmission of sensations continuously to the brain; it cannot be looked

upon as a long sensory tract. The reason for this lies in its extent at various levels of the cord. If it were a long tract of transmission, we should expect its size to increase steadily from below upward, each succeeding higher level transmitting larger numbers of impulses than those below it. But as a matter of fact, the substantia gelatinosa is more developed in the lumbar region relatively to the size of the cord than it is in the dorsal, or even cervical region; and it is reduced to a very small extent in the upper cervical segment. This hypothesis may, therefore, be discarded.

It seems, therefore, evident that the transmission of sensations through the cord must have for an anatomical basis a series of short neurons, each conveying the impulse from the level of its reception in the cord to a level somewhat higher, where again it is transferred to another neuron, and so on, and on, till it reaches the cortex. This hypothesis is substantiated by the researches of Ramon y Cajal and Lenhossék. They have shown that the majority of the neurons of the cord are short in extent; have numerous collateral off-shoots, so that an impulse starting from any one neuronissent upward and downward to other levels, but is not sent to any very great distance. In a word, the structure of the cord is such as to secure a wide dissemination of single impressions to very many levels.

It only requires a brief reference to the known structure of the cortex to substantiate the assertion that here also there is a perfectly homologous arrangement of sensory terminations. The sensory expansions in the cortex are of the widest kind, securing a large diffusion of impulses to an extensive region. In fact, the great rarity of complete anesthesia in one limb from cortical lesions is a striking proof that sensations are diffused over the cortical regions, and cannot be as accurately or closely located in certain limited areas as motor centers are. This, also, is entirely in accordance with the object of sensation, which is to awaken a variety of responses, including conscious perceptions, voluntary acts, comparison of associated im-

pressions, and a train of thought. Is it not, therefore, evident that in the transmission of sensation through the spinal cord, cerebral axis and brain, we have to consider the tracts as different in their structure from those conveying motor impulses, and as consisting of a series of short neurons, closely connected, but with very numerous and very widespread connections? Is it not probable that the result of this arrangement is to secure a diffusion of single sensations to various reflex, vasomotor, trophic and automatic mechanisms, as well as to the organ of conscious perception? And is it not also evident that this view of sensory diffusion explains the apparent inconsistency at present prevailing between the clinical symptoms observed in Brown-Sequard's paralysis and the pathological degenerations occurring in that disease, viz.: the anæsthesia opposite to the lesion, and the ascending degenerations on the side of the lesion? The diffused sensations traverse the uninjured half of the cord, being obstructed at the lesion, and lead to a confusion of impressions and to hallucinatory mistakes of conscious perception. The same thing is observed in the symptom of allochiria.

It seems to me, therefore, that it is useless to search for sensory tracts in the cord as units, or for long sensory columns like the pyramidal motor tracts. The only long columns conducting upward are those related to the cerebellum, and conveying the very numerous sensations necessary for its guidance in maintaining equilibrium and balance; sensations which are necessarily so numerous, and come simultaneously from so many sources that it requires a large central organ to receive and combine them all, with definite tracts leading to this organ.

I should, therefore, conclude that in the conduction of sensory impulses to the brain cortex a large number of different neurons are concerned, which are located in the gray matter and in all the various columns of the spinal cord, and that there are no true sensory columns or long continuous sensory tracts between the spinal cord and the cerebral cortex.

A CASE OF PURULENT, PRIMARY, SPINAL LEPTOMENINGITIS.

By FRANK R. FRY, M.D., St. Louis.

John D——, aged 27 years, an American and a house servant.

I first saw the patient December 10th, 1895. He was in bed, complaining of a severe pain in the lower portion of the back, and general malaise. I did not suspect disease of the spinal cord or membranes. Owing to my absence from the city, I did not see the case again until five days later, Dec. 15th. The record of these five days is, therefore, very imperfect. The salient points obtained, however, are as follows: He had been engaged at his usual duties, inside and outside work about the house, until the day before I saw him. The pain in the lower dorsal and lumbar regions became very severe on the evening of the 10th, after I had seen him. It remained so on the 11th and 12th. On the 14th it was not so severe, but radiated more into the abdomen and thighs. During all of this time he slept very little, and at times complained considerably; but, not wishing to annoy his employers, he evidently suffered much in silence. On the 14th he was seen by Dr. M. A. Bliss, who only made a cursory examination and used a catheter, drawing a large quantity of urine.

When I saw him, on the 15th, the bladder was full, and the catheter was again used. There were paresis and partial anesthesia of the lower extremities, but he was still able to get into and out of bed unassisted. Arrangements were made to send him to a hospital.

On Dec. 16th he was removed to St. Luke's Hospital. At 7 P.M. the following note was made: He complains every few minutes of severe pain seizures, which encircle the

thorax at about the level of the nipples. Complete paralysis and anesthesia of the lower extremities are noted. On closer examination the line of partial anesthesia is found to reach within four inches of the nipples. The patient is comparatively comfortable with morphine given hypodermically.

Dec. 17th. The patient's condition much the same as on the 16th. Although eased by morphine he complains frequently of pain in the back and chest. There is neither stiffness nor tenderness in the neck.

Dec. 18th. Complaining of some soreness and pain in the arms and back of the neck. The muscles of the neck are firm and tender to pressure. Passive movements of the head are resisted. He lies with the arms abducted and the hands resting on the sides of the thorax. He has vomited frequently during the day.

Dec. 19th. The neck is very stiff. He does not move his head, and makes much outcry when it is moved passively. The upper extremities are rapidly becoming paretic and numb. Their position is changed frequently by the nurses at his request. Most of the time he prefers to have them extended on pillows. He is very weak, but not in great pain, except when he is moved in attending to the decubitus, etc. The nausea is controlled by carbolic acid and cocaine.

Dec. 20th. The paralysis and anesthesia are extending in the upper extremities. The neck is very stiff and the head is fixed. The respiration is bad, and the patient very weak. The nausea is still controlled by carbolic acid, etc.

Dec. 21st. On the morning of the 21st the patient is still able to move the fingers and wrist of the right side; the left arm and hand are completely paralyzed. He is failing rapidly during the day. The respiration is shallow and labored. Nausea is frequent. The sensorium is clear. Death occurred at 11 P.M., from exhaustion and respiratory failure.

Subjoined is a record of the temperature, pulse and

respiration. An examination of the hospital record in detail shows that the fluctuations in temperature here noted were not due to the treatment employed. After the patient reached the hospital an attempt was made to keep ice-bags to the spine, but they occasioned so much annoyance to him that they were discontinued after a few hours. Quinine in 3 gr. doses was given irregularly, having been frequently discontinued on account of nausea. Morphine was used hypodermically, and small doses of carbolic acid and cocaine were given to control vomiting.

		Temperature.	Pulse.	Respiration.
Dec. 16th—	7 P.M.	100.6	94	24
Dec. 17th—	1 A.M.	101.6	98	24
"	— 6 A.M.	101.8	102	24
"	— 12 M.	103.2	100	36
"	— 6 P.M.	104.4	120	32
"	— 12 P.M.	102.8	130	36
Dec. 18th—	4 A.M.	104.6	132	38
"	— 6 A.M.	102.6	124	30
"	— 12 M.	99.6	102	30
"	— 3 P.M.	101.2	114	26
"	— 9 P.M.	101.8	108	28
"	— 12 P.M.	100.2	108	26
Dec. 19th—	3 A.M.	99.8	98	24
"	— 6 A.M.	100.2	118	24
"	— 12 M.	100.6	—	—
"	— 3 P.M.	100.8	110	30
"	— 6 P.M.	101.2	112	30
"	— 12 M.	100.2	98	28
Dec. 20th—	2 A.M.	102.8	110	30
"	— 8 A.M.	104.2	108	30
"	— 10 A.M.	102.2	98	24
"	— 1.30 P.M.	101.8	92	22
"	— 4.30 P.M.	101.8	92	22
"	— 7.30 P.M.	100.8	90	26
"	— 12 M.	102.4	86	28
Dec. 21st—	3 A.M.	103.6	92	26
"	— 6 A.M.	103.4	110	32
"	— 9 A.M.	104.2	108	32
"	— 12 M.	103.2	108	38
"	— 3 P.M.	101.6	100	28
"	— 7 P.M.	101.2	98	30
"	— 11 P.M.—death.			

A remarkable feature of this case was the absence of cerebral symptoms. There were none throughout its course, save on the last day, when there were moments of transient delirium or slight wanderings. The mind was clear within an hour of death. The patient endured his

suffering with astonishing fortitude, and when told by his friends on the last day that he could not recover, he simply asked to be made as comfortable as possible until the end, hoping it would speedily come.

A post-mortem examination was made on Dec. 22d. The external surface of the spinal dura presented at no point an inflamed or purulent aspect. At one point in the lower dorsal region there was considerable creamy pus on the outside of the dura. It had evidently oozed through a small hole accidentally made. When wiped away, the dura under it looked normal as elsewhere. On slitting up the dura the cord was found incased in a mass of lymph, mostly of a gelatinous consistence, but at one point, in the lower dorsal region, creamy in appearance. The exudate was greatest in the lumbar and lower dorsal regions, shading off towards the cervical region, where the amount of it was not so great. At many places it was adherent in long strips to the inner surface of the dura. At one point in the lower dorsal region the cord was soft and attenuated for the space of about half an inch. The exudate was more abundant here than elsewhere, and creamy.

After removal of the cord and membranes, the spinal canal was carefully examined. No point of infection or of disease of any kind could be found in or about it. The genito-urinary tract was examined, and in turn all of the abdominal and thoracic viscera, and the walls of these cavities, without finding a pus-focus or disease of any kind. The brain was not examined.

Immediately after opening the dura, six tubes containing a sterilized culture medium were inoculated from different localities along the cord, and turned over to Dr. Amand Ravold, for bacteriological examination. His report is as follows: "Received from you, Dec. 22d, 1895, six tubes containing glycerine agar-agar, that had been inoculated by you with material from a post-mortem. They were placed in an incubator at 36.5 degrees C. After

several days distinct growths were found in five of them. One remained sterile. The growths were plated in agar-agar, and the colonies that formed were separated in pure cultures and studied. One tube showed a pure culture of the streptococcus pyogenes; two tubes mixed cultures of the streptococcus pyogenes and the staphylococcus pyogenes aureus and albus; the remaining two tubes mixed cultures of the staphylococcus aureus and albus. In one of the latter tubes a short, thick, non-liquefying, non-pathogenic bacillus was found, which formed a reddish-brown growth on the medium. It was unknown to me, and was probably an accidental contamination from the air." (Signed) Amand Ravold.

From the patient and his employers we learned that the former had been having a series of boils on the back of his neck. An inspection of the body, both before and after death, showed no evidence of active furunculosis. On the back of the neck were a number of small spots of slightly bluish discoloration, smooth and clean, appearing to have been the sites of small furunculi, which had been healed for some time. We found no cause of the meningitis save the furunculosis, and this it seemed to have followed somewhat remotely.

The cord was put in Mueller's fluid and given to Dr. W. N. Beggs, of Denver, Colorado (formerly of St. Louis), for examination. His report is as follows:

"The inner surface of the dura has, in the preserved specimen, numerous elevated patches of a dark brown color and of irregular shape and size. These are very numerous, and are to be found in various regions without regularity of distribution or arrangement. They may be easily stripped off from the surface of the dura. Other portions of the dura present in the preserved specimen no macroscopically recognizable pathological characteristics, but in sections are readily seen under the microscope to be abnormal.

"Sections of the above-mentioned patches show them

to be composed of a varying admixture of small, round cells, many of them polynuclear or with polymorphous nuclei (leucocytes, pus corpuscles), and a mass of fine homogeneous fibres (fibrin of varying arrangement). In places the latter are collected together into more or less regularly distributed laminæ, with but few of the leucocytes scattered between and through the layers. In other portions the fibres form an interlacing network with strands of varying thickness, forming meshes of the most diverse shapes and sizes. Within these meshes pus cells are present in varying quantities. In other portions the small, round cells are so numerous as to almost entirely conceal the fibrin fibrils. These varying portions pass gradually into each other without any determinate order (fibro-purulent exudate).

"The dura itself also presents a considerable diversity of characteristics. In some parts it is almost normal. In others the innermost layers are almost completely obscured by the great numbers of the small round cells (pus cells) present. In places the cells (both the small round cells and the fixed connective tissue cells) are arranged in rows between the laminæ of the connective tissue fibres after the manner of tendon cells, and in certain parts the fixed connective tissue cells are swollen, the nuclei indistinct, the cell bodies taking up diffusely the nuclear stain, and the contours being indefinite.

"In the pia the blood vessels are widely distended with blood, with, in some of them, a distinctly marginal arrangement of the leucocytes. Here and there foci of hemorrhage of varying degree are to be found. Throughout the meshes of the connective tissue of the membrane, but especially in the immediate neighborhood of the vessels, there is a very considerable infiltration with small round cells. To this is added, in the looser meshes of the membrane, masses of filbrin filaments as already described for the dura.

"The upper portions (cervical and upper dorsal) of

the cord present marked degenerative lesions. These vary from swelling and indistinctness of the component elements of the individual nerve fibres to their entire disappearance. Such changes affecting single or few fibres occur in all tracts of the cord without special localization. In the lateral and posterior tracts especially, but also in a lesser degree in the anterior tracts, the fibres affected are so numerous that they form irregularly-shaped patches of varying size, readily recognizable to the naked eye. Such foci become more abundant toward the upper portion of the cervical region. Their distribution is somewhat irregular, as shown in the accompanying slides and drawings. In many of these foci there are no unaffected fibres left. There is no increase in the neuroglia; these fibres forming simply a loose meshwork, and the interspaces representing the tracts of the nerve fibres formerly occupying them. In the lower portions of the cord I have not found such degenerative changes. The gray substance is normal throughout." (Signed) W. N. Beggs.

PARALYSIS AGITANS AT THIRTY-FOUR YEARS OF AGE, IMMEDIATELY FOLLOWING TYPHOID FEVER.¹

By FRANK R. FRY, M.D., St. Louis.

On November 7th, 1896, I examined E. S.—, a male, aged 37 years. He has paralysis agitans well-defined. The attitude, tremor, gait, speech, facial expression, are all typical. The rhythmic agitation is most marked in the right hand and arm. The left hand and arm are also involved, and to a less extent the legs. The tongue is slightly tremulous. Paresthesia is present, especially in the right hand and forearm, and is sometimes annoying. The patient occasionally has considerable aching between the scapulæ and in the sacral region. There are no other sensory phenomena of moment. The tendon-jerks of the upper and lower extremities are normal, or possibly slightly exaggerated. The intellectual faculties are unimpaired in any particular. The memory, always good, is as reliable as ever. The general condition is fairly comfortable. He has no important complaint, save weakness, *i.e.*, a physical incapacity to engage in his usual pursuit—farming.

From Dr. Jno. H. Ferguson, Mine La Motte, Mo., and the patient, I obtained the following history: The patient had always been a strong, healthy person, until the winter of 1892-3, when he had a severe attack of "grip." This left no marked defects, but possibly some general debility. In August, '93, he was taken ill with typhoid fever, and was confined to his bed four weeks. Two weeks after getting up he was able to go about his farm. Soon thereafter he undertook work which, he noticed at the

¹Read by title.

time, was a little too much for his strength. He was annoyed by a tremor, or shaking, which began during the fever and followed exertion of any kind. Within the next few weeks he remarked that the right arm was distinctly more tremulous than other parts, and that this member was especially weak. He was conscious of considerable tremor towards the end of the fever, *i.e.*, during the last week or ten days of the fever. This, from his description, was the familiar asthenic tremor which frequently follows typhoid, and it seems to have been merged into a paralysis agitans in a peculiar manner. The right arm became more and more inefficient on account of the increasing tremor and weakness. After some months it was apparent that the left arm and hand were beginning to be affected in the same manner as the right. Within the past few months the legs have become involved.

A letter from Dr. Ferguson, March 22d last, states that there has been no important change in the condition during the five months which have elapsed since I saw the patient.

SUBARACHNOID SEROUS EXUDATION PRO- DUCTIVE OF PRESSURE SYMPTOMS, AFTER HEAD INJURIES.

Dr. G. L. Walton read a paper with the above title, and said that in cases offering moderate fibrile movement, prolongation of unconsciousness, and restlessness after trauma, it is of no vital moment whether we adopt the term contusion, bruising or laceration; but when to these symptoms local paralysis is added, the question of operation arises and exact ideas of the pathology become important. Operation over the area indicated sometimes shows only a tense dura, incision of which is followed by a gush of serum. Under the arachnoid membrane the greater part of the cortical cerebrospinal fluid is collected. Serum may be exuded into the subarachnoid space as well as transuded, and sometimes the swollen and congested brain found under the fluid on operation shows that we have to do with an exudation resulting from increased blood pressure, rather than with a compensatory process. Little attention has been paid to this process. The edema of Ballard accompanying hemorrhage is analogous; the serous meningitis of Quincke is analogous if not identical, in so far as he includes external meningitis, but he does not refer to this class of cases. The following cases were mentioned: A boy of six years was struck by a bicycle at noon one day, was restless and drowsy, and on the following day became unconscious, with unilateral paralysis including the face. Operation was considered but postponed. The paralysis had disappeared within four days. A child, three and a half years old, fell from a swing, striking the head; was drowsy, and the next day one arm was paralyzed. The paralysis began to lessen on the third day, and rapidly disappeared. A young woman fell, striking the head violently. Unconsciousness, restlessness, vomiting, and hemiplegia appeared. Operation revealed a tense dura, incision of which was followed by a gush of serum. There was relief of pressure symptoms, but death ensued from the underlying condition. His conclusions were: 1. A severe blow may result in local bruising and congestion with subarachnoid serous exudation. 2. The fluid may be imprisoned and cause focal paralysis. 3. The process is not compen-

satory, and is allied to the serous meningitis of Quinke. 4. The lesion is self-limiting. 5. Diagnosis from hemorrhage is difficult. Atypical course, absence of steady increase of symptoms, and persistence of sensitiveness point to serous exudation. 6. Immediate operation is not necessarily indicated in focal paralysis, though, perhaps, always justifiable. 7. This condition is specially to be borne in mind before operating on children and young adults.

DISCUSSION.

Dr. Wm. N. Bullard—Two years or more ago, in a paper read before the Surgical Section of the Suffolk District Medical Society, and later printed in the Reports of the Boston City Hospital, I drew attention to the existence of increased intracranial and intradural pressure in cases of injury of the head. Further investigation and study of these cases has confirmed my belief that this condition is the pathological cause of many of the symptoms arising in these cases, and that the presence and degree of this increased intradural pressure is one of the most important factors in determining whether or not operation should be performed. I think that it may now be received as an accepted fact that in many cases, at least, of injuries of the head such an increase of intradural pressure exists. That this intradural pressure is due in part in some cases to effusion into the spaces between the meninges, and possibly into the tissues of the meninges themselves, is unquestionable. But it is yet undecided how far this effusion may be considered as the principal cause of the increase of intradural pressure, and how far a soaking of the brain tissue and a filling and enlargement of the intracranial vessels, especially those in the brain, contributes to this result. Certain it is that in many cases where, after injury, there is a marked intradural pressure, the opening of the dura does not let out any large amount of serous fluid.

As to how far the symptoms due to this increased pressure within the dura, when localized in the form of paralysis, hemiplegia or some other type, are to be considered as indications for surgical interference has not yet been fully determined. We know, however, that patients, who after injury to the head become gradually unconscious and shortly develop a hemiplegia, having, in short, such symptoms as would suggest a hemorrhage within the cranium, may recover spontaneously without treatment within a few hours. This is apparently more frequently the case in children than in adults. I would especially call attention in this relation to the case of Dr. Prince reported last winter.

Permit me, finally, to present my regrets at not being able

to attend this meeting and to give my thanks to Dr. Walton for giving me a summary of his valuable paper. I believe this to be a most important subject, and that in its thorough knowledge and consideration lies the key to proper discrimination in operation in head injuries.

Dr. Morton Prince, of Boston—I think the theory of Dr. Walton is very plausible, but difficult of substantiation. I remember the case of a child who had a fall, and showed the classical symptoms of meningeal hemorrhage, such as unconsciousness following an interval of consciousness, hemiplegia, etc. Operation was deferred, and the child was perfectly well on the following day. The most plausible theory in this case is, I think, that of local edema.

Dr. B. Sachs, of New York—The theory advanced is not a new one. After head injuries three possibilities are to be considered: hemorrhage, local edema, and purulent meningitis. Dr. Walton's view is natural and plausible, and I agree with his views as to differential diagnosis. I would advise deferring operation until there was positive proof of serious damage to the brain, at any rate until after the lapse of forty-eight hours or several days.

Dr. J. J. Putnam, of Boston—I assisted at an autopsy on a woman who, during convalescence from a severe influenza, had developed localized twitching of arm, leg and face every twenty minutes, followed twenty-four hours later by hemiplegia and rise of temperature. There were only edema and a wet, soft brain, and there was no local accumulation under the pia. I think the convulsions and hemiplegia were due to the general edema. The precise cause of the nerve irritation in these cases is not clear.

Dr. Hugh T. Patrick, of Chicago—I have had a case communicated to me almost exactly like that of Dr. Prince. A child received a fall, which was not severe, in the latter part of the afternoon, after which he played about as usual. At the supper table he suddenly let fall a fork from his right hand, and soon thereafter had a one-sided fit. Following this he became stuporous, and when seen, several hours later, was almost comatose; but a distinct hemiplegia was made out. Operation was declined by the family, and two days later the child was absolutely well. A localized edema would seem to be the only possible explanation of such an occurrence.

Dr. C. A. Herter, of New York—I believe that local effusion can give rise to these symptoms. I have observed a case of extreme general edema of the arachnoid following syphilis. There was some doubt as to whether the fluid was a simple effusion or an exudate; the inflammatory process, however, could hardly occur so rapidly. The symptoms may possibly be due to cerebral anemia, dependent on compression of the

brain by the fluid. Pronounced general edema of the brain in children may be accompanied by any cerebral symptoms.

Dr. Joseph Collins reported the following case as an example of post-traumatic, circumscribed, serous meningitis, with paralytic symptoms due to pressure:

A man, about fifty years old, was admitted to the City Hospital in the wards of Dr. Brewer, with whom I saw him. His history, which was obtained from Gouveneur Hospital, to which he had been taken immediately after the accident, was that he had fallen from a hay-loft; in falling had struck his head, and had received an open wound of the scalp over the left temperoparietal region. This, aside from a moderate degree of shock, seemed to be the only effect of the accident. The wound was dressed, and the patient received otherwise no special attention. He soon became stupid, slightly irrational, and complained, at times, of headache. He was transferred to the City Hospital, after the scalp wound had healed, and examination at this time showed:

1. Mental sluggishness; slowness in answering questions—frequently the patient would stop in the middle of an answer and not finish it; a dazed, non-attentive, dreamy, uncommunicative state; an absence of demands of any kind, but response to the calls of nature.

2. Paralysis of the right upper extremity, principally of the shoulder, reminding one very much of a "deltoid" paralysis; the possibility of movement of the fingers and hand, and of flexion and extension of the forearm in a limited degree.

3. Temperature slightly elevated; respirations sometimes slow, sometimes rather accelerated.

4. Slight stiffness of the neck in varying degree.

5. Pupils of moderate size, equal and of slight response either to light or in accommodation. It is probable that they did not react in accommodation.

6. Absence of cranial nerve involvement, so far as could be made out.

7. Inability to stand or walk.

8. Knee jerks elicitable, but both very weak, and the right more so than the left.

9. Necessity of catheterization. Later there was some dribbling.

The symptoms became aggravated; the mental obtundity more profound; the paralysis of the right upper extremity more complete; the stiffness of the neck less marked; and the impairment of vital functions more evident. The patient was unable to make any response, and the paralysis extended to the right leg.

A diagnosis of intrapial hemorrhage or edema, situated over the lower end of the Rolandic region of the left hemi-

sphere, was made, and the surgeon was advised to operate over the shoulder centre of the left hemisphere. The operation was undertaken, and on removal of a flap of bone from the skull over the left Rolandic region, the dura was ruptured, and about three ounces of clear fluid gushed out. On examination it was seen that the pia was adherent to the dura and the dura slightly adherent to the cranium, but sufficiently to cause rupture when the bone flap was pulled back. After the fluid had escaped there seemed to be a profound concavity in the surface of the hemisphere corresponding to the seat of the exudation. The finger introduced into the skull opening, and passed along the parietal pia, failed to detect any further abnormality, and the wound was closed.

The patient withstood the operation very well, but there was no improvement in his general condition, and he died five days later.

The autopsical findings were unlike anything previously encountered. The only departure from normal within the cranial cavity, with the exception of a comparatively slight degree of ventricular distension, was the deep circumscribed concavity of the lower Rolandic area of the left hemisphere already spoken of. It was impossible to find the reason for the limitation of the cavity, as no membrane could be found. The limiting membrane had been broken down, probably at the time the surgeon swept his finger around the concavity in search of further abnormalities. The gyri were flattened and sunken in, and the fissures were smoothed out in this concave area, and on section through the gyri involved the cortex was seen to be very much thinned. The meninges over the rest of the brain were quite normal in appearance. Microscopical sections have yet to be made.

The pathogenesis of this localized, circumscribed, parietal, pial edema is probably the following: After the injury a low grade of serous meningitis developed, and at the borders of this area of meningitis a slight amount of plastic exudate was formed, which was sufficient to unite the two layers of the pia and to give the exudation definite limitation. This limiting wall was not found on necropsical examination, because in removing the dura, to which in all likelihood the pia was somewhat adherent, it was torn away or broken down, and it is not improbable that future microscopical examination of the pia will show the remains of this limiting wall.

The absence of indications of irritation referable to the motor area in the beginning of the patient's disorder, and the presence of meningeal symptoms, particularly the psychical ones, had much to do in deciding the diagnosis and the decision of advisability of operation.

In response to an inquiry, Dr. Walton stated that the tem-

perature in these cases is generally only a moderately elevated one, as high as 102 degrees; such as is not always observed in simple hemorrhage.

Dr. J. J. Putnam—I saw an extremely interesting case some years ago, of localized hemorrhage from the veins of the pia mater, where a dense, thick clot eventually formed, causing symptoms, first of irritation, finally of paralysis and coma. If such a clot can be confined without the production of inflammation, within comparatively close limits, I do not see why the same thing might not be true of edema.

The President—The condition of localized edema should not be a question of theory, but a question of fact, and I think that we have testimony enough before us to-day to warrant the conclusion that such a thing is possible—indeed, not only possible, but frequent. I have seen this condition in three cases that I recall where operations have been undertaken, and where a distinct condition of subarachnoid edema, not in any respect inflammatory, was present, and subsided—could be watched in its subsidence—during the operation; returning subsequently to the operation—in one case two days after the operation—and then was again relieved under dressing; each time, at the period of the return, producing definite symptoms. So that in the living subject I believe we have proof of localized edema of the arachnoid and localized edema of the brain.

In a post-mortem examination recently made in connection with Dr. Peterson in a case of brain tumor, where the tumor was not of large size, both Dr. Peterson and myself were impressed by the large amount of edema of the entire hemisphere in which the tumor lay. The edema was so great as to increase the measurements of the hemisphere certainly by an inch, and yet the tumor itself was very small, there being therefore a very distinct edema of very marked character around the tumor. Nervous pathology should come into line with pathology in other parts of the body, and there is no reason why edema should not occur in connection with tumors and small lesions in the brain as well as in other parts of the body. Dr. Janeway, speaking of this particular case, called to mind two cases in which he had seen coma followed by apparent recovery, and then coma and death, in which the only explanation of such alternation in the symptoms in a condition of brain tumor, where the tumor was found at the time of autopsy, was to be found in the existence of a transient edema, which had disappeared and then returned. I think this discussion has been of considerable interest, and we are indebted to Dr. Walton for bringing the subject before us.

IDIOPATHIC INTERNAL HYDROCEPHALUS (SEROUS MENINGITIS) IN THE ADULT, WITH REPORTS OF THREE CASES (TWO WITH AUTOPSIES).*

By MORTON PRINCE, M.D.,

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FOR our clinical knowledge of serous meningitis, or primary idiopathic hydrocephalus, in adults, we are chiefly indebted to Quincke. Before Quincke's monograph¹ several cases had been reported by different writers (Huegenin, Eichorst,² Oppenheim, Annuske), and the later text-books contain brief references to, or descriptions of, the affection. Eichorst's paper was a valuable contribution, but all the later accounts are for the most part based on Quincke's original study. Most are very meagre and insufficient, and give a very inexact picture of the disease. Oppenheim, however, has given in his text-book a very clear account of the affection. In a later paper³ Quincke has reported ten additional cases, and makes a further contribution to the subject. But even Quincke's description will probably have to be modified in the future, as many of his cases never came to autopsy, and therefore the diagnosis in these cases must be open to suspicion. Post-mortem findings have been obtained, however, in enough cases to establish the disease and its chief clinical manifestations.

* Printed in the Medical and Surgical Reports of the Boston City Hospital, 1897.

¹ Volkmann's Sammlung, 1893, No. 67.

² Zeitschrift f. lin. Med., 1891, 19, p. 181.

³ Deutsche Zeitschrift f. Nervenheilkunde, 1896, ix, p. 149.

The pathology of the disease must still, however, be regarded as obscure, and invites further investigation. That the disease process is to be regarded as an inflammation, seems questionable in light of the findings in many of the cases. In one of my cases, for example, even a microscopical examination failed to reveal what are usually regarded as evidences of inflammation. The affection is probably of frequent occurrence, and in the future is certain to be more often recognized after we have learned to be familiar with its clinical features.

On October 12th last, I was asked by Dr. J. C. Munro to see a patient in the surgical wards of the City Hospital. The patient, a woman, had been admitted to the hospital about four weeks previously on account of injury to the head. She had been kicked about the face and head by her husband. This was about four o'clock P.M., and she was said to have been unconscious until about midnight of that day. The next day (September 15th) she was admitted. On entrance the records state "much swelling of left face; whole left face bruised and ecchymosed; some ecchymosis on left side of neck; slight abrasion of left arm and chest; both eyes blackened; marked tenderness over face and entire head; no sign of paralysis." T.: 100.

During the following four weeks (until October 11th) she had exhibited mostly symptoms of a puzzling character; among them irregular but high fever, mild chills, severe headache, obstinate vomiting and pain in the back of the neck were prominent, but not continuous or co-existing. At first there had been delirium, but later her mind seems to have been clear, until the fourth week. Unfortunately the hospital records are not as full as could be wished, in view of the future events, but a study of them with the additional information derived from those in attendance on the case shows the conditions to have been substantially as follows: The above general symptoms were most marked during the first six days; after that improvement set in, and during the following three weeks

her condition was fairly comfortable, and, excepting that the fever continued, and at one time one symptom was prominent and at another time another, she might have been thought to be convalescing; but there was a steady and gradual failure of strength. Headache and vomiting were present at times, as well as pain in the back of the neck.

Dr. Bullard saw the patient the day after entrance, and found no objective symptoms of injury to the brain; tenderness of the head and face, headache and vomiting were the most prominent symptoms. T.: A.M. 104.2; P.M. 104.5.

September 17th. (Third day.) "Temperature has dropped about three degrees. Patient complains chiefly of headache and also of general pain. The vomiting continues. Was quite noisy and delirious last night. . Seems mentally clear to-day." T.: A.M. 102; P.M. 100.8.

Fourth day. "Patient improving, for the temperature is dropping. The headache is less severe, and though there is still some vomiting, patient says she feels better." T.: A.M. 98.6; P.M. 99.8.

Fifth day. "Patient again shows a high temperature, but the subjective and objective symptoms are no worse. Still complains of headache. Vomits quite often." T.: A.M. 104.5; P.M. 104.5.

Seventh day. The headache and vomiting have subsided, but the patient complains chiefly of *pain* in upper back and in the *back part of neck*. "Diarrhea for the past two days." Temperature still up, 105°.

On the 26th (eleventh day), all the objective symptoms had ceased. Dr. Bullard, who again saw the patient, was unable to find evidences of cerebral disease, and suggested, in view of the exacerbations of temperature and chills, the possibility of malaria, and advised quinine. (This gives an idea of the negative character of the symptoms at this time.)

Three days later Dr. V. Y. Bowditch saw the patient, but was unable to make a diagnosis. Mental symptoms had

again returned, but during the following thirteen days the patient continued fairly well so far as local symptoms were concerned, though in general strength she was going downhill. At one time pain in the back of the neck, at another time headache, at another vomiting, were prominent. During the latter week of this period she became noisy at night. It was now learned that for some time before entrance to hospital her actions had been peculiar. She had "had freaks of leaving home suddenly without apparent reason, and returning after some time." At the end of the fourth week (October 11th) local nervous symptoms of importance were noted for the first time, as follows: "Condition has not changed materially until to-day. This morning right pupil was found to be dilated and the left contracted. Patient had a convulsion of the right arm, which began in the thumb and ran upward. Had some twitching of the left arm also. Continued to have twitchings in both arms all day. Some twitchings of the right side of face. Complains of severe pains in back of neck. For the past week has been noisy at night; seems rather dull and stupid to-day."

October 12th. "Slight twitchings of both arms and of the right face continue. Right pupil *less* dilated. Still complains of pain in the back of neck." In the afternoon of this day I first saw the patient. She was dull and apathetic; so much so that when testing the ocular muscles it was with some difficulty that I could induce her to follow the finger with her eyes, or that I could examine the field of vision. The right pupil was dilated and stable. Left pupil slightly dilated and stable (third nerve). Paralysis of the right external rectus (sixth nerve).

"There is marked rigidity of the muscles of the back of the neck as well as tenderness of this region. Any attempt to move the head causes considerable pain about the upper cervical vertebræ. Pressure over the vertebræ is likewise painful. She lies with a pad under the neck for

relief. Protrudes tongue straight." (I thought there was slight paresis of the right lower facial muscles, but, as this was not observed the next day, I thought this must have been an erroneous observation. In the light of future events I now think this observation was probably correct.) No anesthesia. Nothing remarkable about reflexes. No paralysis of the arms nor legs, but the arms seem to show more resistance to passive motion than normal; a sort of spastic or rigid condition by which attempts to suddenly flex and extend the joints induce contraction of the muscles. This is not marked, but it is unmistakable.

October 13th. House officer reports that in early morning patient mentally much more dull, etc. To my surprise, at visit (about 11 A.M.) patient is comparatively bright; mind fairly clear; laughs; apparently natural, but speaks of an imaginary visit which she says she has made while in the hospital. When asked if she really made the visit or if she dreamed it, she considers for a moment and then says that she dreamed it. Memory fair, but does not remember origin of illness, etc. Still mind not normal. Temperature normal. Abdomen retracted and firm. The paretic condition of ocular muscles is more marked. Right internal rectus is paretic, and the eye does not move downwards as well as the left. Field of vision tested by finger, normal.

October 14th. Change again for the worse; moderate rise of temperature. Mind much more dull and stupid, even than on the 12th. Ocular paralysis more marked still. "Ptosis, right eye. General condition worse, evidently failing. (*No optic neuritis*. Media and fundi oculorum normal (Dr. Bossidy). Examination of ears by Dr. Leland revealed nothing abnormal.)" It seemed evident that the patient was going to die.

On analyzing the symptoms of this case for the purposes of diagnosis it was evident that we had to do with a cerebral disease which appeared to be the consequence of injury. The symptoms were local and general.

The general symptoms which had been noticeable during the course of the disease were: irregular fever, slight chills, headache, vomiting, stupor, delirium.

The local symptoms were: 1, cervical rigidity, tenderness and pain; 2, paralysis of the right sixth nerve; 3, paralysis of right third nerve; 4, paralysis of both pupils; 5, retracted abdomen; 6, mild rigidity of both biceps (arms); 7, localized spasms; 8, temporary weakness of right seventh nerve.

It is noticeable that the local symptoms were latent; that is, that they did not develop until the fourth week of the illness, and that up to that time there was nothing that directed special attention to the brain as the seat of the trouble aside from the fact of the injury to the head.

The general symptoms also showed a peculiarity that should attract attention, although I did not give the weight to it that I should have done, viz., there was a great variability in their intensity (at one time one symptom being the most prominent, at another time another), while the general condition of the patient, with the exception of the temperature, showed a tendency to improvement, followed by relapse. This variability was marked even during the final relapse, after the local symptoms have developed, when the patient was comatose or delirious at one time, and clear in mind at another. The temperature was very irregular, being at times as high as 103° - 105° , at other times normal. The temperature taken every four hours showed a similar irregularity.

In making the diagnosis that I did, I was led by the following considerations and reasoning; the history indicated that we had to do with a traumatic affection; the general symptoms (chills, fever, headache, vomiting, delirium, stupor), looked at *aside from their course*, were compatible with meningitis, sinus thrombosis, abscess, and, possibly, with cerebral laceration; the local symptoms (paralysis, spasms, cervical rigidity, etc.), by themselves, were strongly indicative of meningitis of the base.

But it seemed extremely improbable that such a meningitis, presumably purulent, should continue for four weeks without producing local symptoms; in fact, should run a latent course, during which a general improvement should occur, and then end with a sudden exacerbation and development of paralytic symptoms of the basal nerves.

Sinus thrombosis could be excluded by the absence of middle ear disease, injury to the skull, and all evidence of septic infection.

Cerebral laceration was incompatible with the course of the disease.

On the other hand, it seemed to me that the symptoms were compatible with, and explainable by, abscess of the brain. Trauma is known to cause abscess, which also may run a latent course. It seemed to me at the time, that the general symptoms were explicable by a latent abscess, which finally, by pressure, or possibly by rupturing and thereby exciting a meningitis of the base, had caused the local symptoms. The strongest point against this theory was the temperature, which in latent abscess is said to be usually normal or sub-normal; but a study of recorded cases showed that this statement is too general and that fever is not uncommon. Besides, in fact, fever is also uncommon in acute hydrocephalus or serous meningitis.

For reasons unnecessary now to go into, the most likely location of such an abscess seemed to be, first, the cerebellum, and next, the temporo-sphenoidal lobe. Accordingly, while recognizing the uncertainty of the diagnosis, I felt justified in advising an exploratory opening. On the 25th, Dr. Munro operated, exploring first the cerebellum, and then the base, with negative results. Not finding any pus in the cerebellum or about the base, it was decided to make a second opening over the temporo-sphenoidal lobe, but before this was completed it became evident that the patient was bearing the operation badly, and it was thought best not to go farther.

The patient only partially regained consciousness after

the operation, and continued to fail. Death took place on the early morning of the 18th, the third day after the operation.

Autopsy, Oct. 19th, 1896, 32 hours post-mortem, by Dr. F. W. Draper.

The body was that of a woman 29 years old, greatly emaciated.

There were scarcely visible remains of ecchymoses in the left temporal region, without definite outlines, and yellowish in color, with a bluish tinge.

The left pupil was larger than the right.

The left temporal muscle showed a slight degree of infiltration with blood.

The skull was not fractured and showed no injury whatever, except the recent surgical lesions.

The dura was normal.

The surface of the brain at the vertex showed complete obliteration of the sulci and flattening of the convolutions, with injection of the vessels of the pia; the cerebral face was dry and sticky, but not inflamed.

On palpation of the brain laterally there was abnormal elasticity.

At the base there was more than the normal amount of cerebro-spinal fluid, with a moist and sodden state of the brain tissues exposed.

The vessels at the base were normal.

There was no pus in the arachnoid spaces.

Section through both of the lateral ventricles found those cavities dilated to about three times their normal capacity; they contained clear fluid with a yellowish tinge to the amount of five fluid ounces.

The ependyma was pale, slightly swollen, velvety, showing a sodden aspect.

The velum interpositum was pale, swollen and moist.

There was no appearance of injury, old or recent, on section of the cerebrum.

The brain substance was moist, but not injected.

The heart, lungs, spleen, stomach, pancreas, intestines, liver, gall-bladder, kidneys, supra-renal bodies, great vessels, womb, bladder and ovaries were normal.

Case II.—C. G., housewife, aged 30. Previous history given by Dr. R. C. Macdonald, under whose constant care she was from August, 1888. Well and strong until second confinement, in 1891; never well since then. A pronounced neurasthenic "of that unsatisfactory kind that nothing seems to help;" sometimes better, sometimes worse. Chief suffering of menstrual periods; that is, attacks of prostration, vomiting, headache, abdominal pain and great tenderness, extreme wakefulness and nervousness. Sometimes slight chills and slight fever; no stupor nor delirium. Between attacks, fairly well. Attacks grew progressively worse, and lasted about two weeks. During last year vomiting was a prominent symptom. Neurasthenic and other symptoms were thought to be due to ovarian disease, and ovariectomy advised. (At the autopsy cysts were found.) Dec. 10th, 1896, well, excepting for a cold. December 19th, one of her menstrual attacks.

December 26th, headache began; said to have been quite well since December 19th. On December 29th reported better, excepting for severe headache, but not seen until January 2d; then chills; temperature, 103; headache, vomiting, etc. Diagnosis: Suppressed menstruation, peritonitis, subacute and general inflammation of the pelvic organs. Admitted to Boston City Hospital, Jan. 5, 1897; service of Dr. A. L. Mason. Patient complained, previous to admission, of chills of an indefinite intensity and character. Condition on entrance: Complaints of *pains* all over, and especially in the head and neck; frontal and occipital headache; also an area of pain along the sagittal suture; neck is very sore, and movement of the head causes pain. Some, but not localized, pain in the abdomen, also in genitalia and across chest. Physical examination: Organs negative; knee-jerks much diminished; no ankle clonus; no edema nor anesthesia; *tenderness of the muscles of the neck, with tendency to retraction*. Temperature, 100; pulse, 80.

Continuation of this condition for about twelve days. Examination of the blood for malarial organism, negative. *Slight optic neuritis*; no otitis. During the first two weeks after admission the temperature was very irregular, the highest point reached being $103\frac{1}{2}$, while at times, for short intervals, it was normal. After the first two weeks, temperature was normal or subnormal. Urine at first normal, but at one time later contained one-half per cent. albumin, blood and casts. Thirteenth day after admission, change for the worse; comatose; involuntary movements of the bowels and bladder. This condition persisted during the following six days, when Klebs-Löffler bacillus was found in the throat without inflammation. Patient removed to the contagious wards (South Department) to care of Dr. McCollom.

Records of this department state condition on admission as follows: Pulse slow, regular, good volume and tension; pupils equal and react normally; no retraction of the head; slight tenderness over nape of neck; intelligence stupid; heart, lungs and throat negative; decided muscular weakness. For a short time after admission patient was obliged to be restrained to be kept from falling out of bed, which she did once or twice. Temperature slightly subnormal during first three weeks, after that normal. Pulse varied between 60 and 90 degrees.

Ten days later it was noted that the facial expression was that of a dement. Facial muscles smoothed out; pupils became dilated and reacted sluggishly to light and accommodation. During the following four weeks patient remained in the same condition of dementia. The coma had passed away, but the intelligence varied; sometimes the patient seemed brighter, and at other times more demented; occasionally some delirium. A good deal of nausea and vomiting, and more or less headache. But excepting condition of the pupils, no focal symptoms; no paralysis, nor anesthesia, nor tremor, but general muscular weakness. Speech, muscular co-ordination and reflexes

normal. On several occasions the house officer was called at night because of the poor condition of the pulse; in a few minutes it would become of fair strength; these spells similar to the one before her death.

March 4th. No special change, save that for the last few days has complained of constant frontal headache; says, "I believe my head will burst." Some vomiting. Temperature subnormal for the past six days; pulse about 60. In the afternoon an attack in which the pulse became several times alternately almost imperceptible and of fair strength. Half an hour later, death. Duration of illness about eleven weeks.

From the above it will be seen that the prominent symptoms in this case were: Impairment of consciousness, viz., delirium, stupor, coma and dementia; the last simulating, as was thought at one time, general paralysis. Chills and fever at first, later normal and subnormal temperature; rapid, followed by slow pulse; abdominal pain and tenderness, and cervical pains; severe headache; nausea and vomiting; optic neuritis; muscular weakness, but no paralysis nor anesthesia. Some of these symptoms appeared and disappeared during the course of the disease, which exhibited temporary improvement with relapses. The demented facial expression, a part of the general muscular weakness, and the absence of spasms, are worth noting. The different diagnoses of different attending physicians give an inkling into the general picture of the disease at different times: "Peritonitis," "Cerebro-spinal Meningitis," "General Paralysis."

Autopsy: Kidneys, weight 300 gms. Very firm, of a dark-red color. Cortex injected. Relation of cortex and pyramids normal.

Both ovaries cystic, cysts varying in size from a small pea to that of a walnut. Bilateral laceration of cervix

Brain: weight, 1095 gms., small. Convolutions flattened markedly over convexity, particularly over frontal lobes; in places practically abolished. No areas of soften-

ing. The pia strips easily, leaving a moist, soft, smooth surface. The gray matter has less color than normal. Brain substance easily torn, clings to knife. The punctæ cruentæ absent. Ventricles contained about 150+cc. of clear serous liquid. Ventricles markedly dilated, particularly the fourth ventricle. Ependyma smooth, shiny, no adhesions nor roughenings anywhere. Brain markings normal, except that basal ganglia are paler than normal. Vessels at base show no sclerotic changes. Cerebellum and medulla moist, otherwise normal.

Cervical and beginning dorsal cord soft, flattened out as cord is laid across finger. Markings less distinct than normal. Other organs not remarkable.

Microscopical examination of the ependyma, made by Dr. J. J. Thomas, showed "no changes beyond a slight increase of the neuroglia lying beneath the ependyma, and no evidence of any inflammatory changes. There is some infiltration about the blood vessels of the brain, and signs of atrophy of the brain."

Anatomical diagnosis — Hydrencephalus with edema of the brain. Hypostatic congestion of lungs and kidneys. Slight arterio-sclerosis. Cystic degeneration of ovaries; bilateral laceration of cervix.

Case III.—The following case, which I saw in consultation with Dr. Henry F. Leonard, probably belongs here, although, as recovery took place, the diagnosis must remain unconfirmed, and rest on exclusion. Mrs. B., middle age, dressmaker. Condition at time of consultation: Semi-comatose; aroused with difficulty; temperature normal or subnormal; no paralysis; incontinence of bladder and bowels; urine normal; heart negative; no pulmonary symptoms; pulse weak. Patient seemed moribund, and an absolutely unfavorable prognosis was given. The history, which it should be said Dr. Leonard has given me from memory, is as follows: *Onset*—well until the day she was taken ill; worked during forenoon, but severe headache, for which she consulted Dr. Leonard. Besides headache,

felt as if she was falling backwards; "eyes were blurred at time of first visit." *Progress*—Headache continued; no vomiting at first, later several attacks; pain in almost every part of the body; screamed when touched or when moved; temperature normal at first, later subnormal; no paralysis, anesthesia nor spasms; urine normal; no cough nor pulmonary symptoms; delirium; muttered much at first, talked to imaginary persons, then coma or stupor; more or less unconscious for three weeks or longer; gradual loss of flesh and strength each day; "ice-cold" extremities; dilated pupils; low respiration; slow pulse; nourished by rectum. "For about two weeks seemed to be on verge of collapse." "During the illness, and for several months after the acute symptoms had subsided, she had a vacant, idiotic expression" (facial paresis?). Gradual recovery. *Duration*—Two months. Fully six months before headache was gone—much longer time before she could work. Eyes so weak that she could not use them for several months.

No other diagnosis than serous meningitis seems possible in this case. The disease was plainly cerebral, and if cerebral, probably a meningitis; the absence of fever, the prolonged course, complete recovery, absence of palsies, render the purulent form unlikely, and point strongly to the serous variety.

In view of the probably frequent occurrence of the affection, its importance, and the very brief references or descriptions that are to be found in English literature, a very short résumé of our knowledge may prove acceptable. The following account is for the most part based on Quincke's monographs:

The fundamental conception pertaining to the affection is that a meningitis, particularly an ependymitis, may give rise to a simple serous effusion, as well as a purulent one; just as an inflammation of the pleura may cause serous effusion into the pleural cavities, and one of the synovial membranes an effusion into the joints. Thus we

may speak of a serous meningitis or a purulent meningitis. Hitherto it has been customary to picture meningitis only in its purulent (or tubercular) form. By bringing meningitis into analogy with inflammation of other membranes (pleuritis, pericarditis) we enlarge our conception of the disease.

As such a meningitis may occur at all ages, and give rise to collections of fluid in the ventricles, we have a primary idiopathic internal hydrocephalus of adults, as well as the classical form of children.

The causes, so far as known, may be regarded as trauma, mental strain, alcoholism, otitis media and acute infectious diseases.

The affection may be either acute or chronic.

The acute form in its symptoms simulates purulent and tubercular meningitis.

The chronic form presents symptoms so closely resembling tumor that it is usually diagnosed as such. According to Quincke, in another variety of the chronic form the symptoms are those of neurasthenia. It may be pointed out, however, that as the conclusive proof of an autopsy is necessarily lacking in such cases, the existence of this neurasthenic form must be open to doubt, and must rest on theoretical considerations. The previous neurasthenic condition in case II. above is strongly suggestive of this view.

The affection is not so rare as would appear from the paucity of reported cases, the acute cases being mistaken for purulent meningitis, and the chronic ones for tumor. Even when at the autopsy an effusion is found it is not customarily regarded as pathologically a sufficient cause of death.

The cases may be further classified as follows:

I. Acute onset.

(a) With acute course.

(b) With chronic course.

II. Chronic course.

(a) With chronic progressive course.

(b) With acute exacerbations.

The symptoms, as in other brain affections, are general and local, and depend largely upon increase of pressure. The general symptoms are headache, vomiting, fever, somnolence or stupor, delirium, convulsions, optic neuritis. The local symptoms are paralysis (especially of the cranial nerves), exophthalmos, cervical pain and rigidity, hyperesthesia, pain in extremities, etc. It is by its course, and greater or less intensity of individual symptoms, that it is to be distinguished from other forms of meningitis and tumor.

When the onset is acute it may resemble purulent meningitis, but more often it is slower as in the tubercular form; but even the latter, in adults, is more rapid and more continuously progressive. Nevertheless, there are cases which are apoplectiform in their onset, with loss of consciousness. The pulse is often slow. Fever is either absent or only slight, of short duration and irregular course. (To this, Case I., above reported, is an exception.) The headache is diffuse, or located in forehead or occiput, of varying intensity and sometimes periodic, or with periodic exacerbations. Usually, some days after the onset, dulness of consciousness is observed. As a rule, this is manifested later than in purulent meningitis. It is only in fatal cases that this is continuous and profound. Usually, like headache, it is of varying intensity and alternates with, or is associated with, unrest, delirium and sleeplessness. Vomiting is common. Rigidity of the neck, with tenderness and pain of motion, may be prominent. (This was marked in Cases I. and II.) Paralysis of the cranial, particularly ocular and facial, nerves may be present, though not usual; that of the sixth is most common, it being most subject to pressure on account of its course. These paralysees are apt to be slight and of varying intensity. The pupils are unequal and react slowly, or are stable. Oppenheim describes weakness of the extremities as common.

Spasms, more or less diffuse, may occur. An important finding is optic neuritis, with atrophy. This is more frequent than in other forms of meningitis. In the chronic cases it is frequently associated with headache, vomiting and mental dulness as the cardinal symptoms, thus simulating tumor; but after weeks or months such cases may end in recovery. The visual defect, instead of blindness, may be that of bi-temporal hemianopsia due to pressure from the dilated third ventricle upon the optic chiasm.

Other symptoms are exophthalmos, hyperesthesia, pains and intention tremor. In Case I. rigidity of the abdomen and arms was present. A demented facial expression was noted in two of the above cases.

The acute cases may, after a course of some weeks, end in complete or incomplete recovery or death, or become chronic. A remaining optic atrophy may be the sole indication of past cerebral disease. The chronic cases may pursue a varying course, with ups and downs, and acute exacerbations. Such an exacerbation may, in the absence of a complete history, give the erroneous idea of an original acute attack. (In Case I. it is a matter of doubt whether the trauma caused such an exacerbation or an original attack.) They may continue for years, ending finally in recovery or in death. For the diagnosis, which at present in most cases must be extremely difficult, Quinke lays stress upon the great variations in the intensity of the symptoms from day to day; at one time one symptom, at another time another coming to the foreground. In the chronic cases the occurrence of remissions and intermissions must largely be relied upon to distinguish them from tumor, and when the not very common focal symptoms (palsies, etc.) are present, the fact that instead of progressively deepening, as with tumor, these things have a temporary or varying existence.

The pathological findings are for the most part limited to the accumulation in the ventricles of clear fluid which does not materially differ from normal. Quinke makes the point that while the effusion into the arachnoid from

cortical meningitis is almost always turbid, and therefore more or less rich in cellular elements and albumin, the reverse is the case in meningitis of the ventricles.

The ventricles may be enormously distended, and the pressure so increased that the convolutions may be flattened and the sulci obliterated in appearance. The changes in the ependyma seem to be limited to hyperemia, in the acute stages, and to some slight thickening and change in texture (described as smooth, velvety, granular, sodden) in more chronic stages. The pia may be hyperemic also, and share somewhat in the process. Quincke, in discussing the pathogenesis, likens the affection to angio-neurotic acute edema of the skin, an analogy which renders intelligible the sudden development and variability of tension (symptoms) observed.

It is not quite clear to my mind that the effusion is to be regarded as of an inflammatory character. Certainly in Case II. above, the condition of the ependyma does not suggest inflammation, and the same may be said of the findings in some of Quincke's cases. The pathology of the effusion seems to me to be obscure and deserving of further research.

As to treatment, Quincke ascribes recoveries to the use of mercury. Lumbar puncture has its advocates. In cases which pursue an unfavorable course and a fatal issue seems imminent, I should be inclined to advise tapping the ventricles, although much might be argued against this procedure on the ground that its effect would be likely to be only temporary.

Case IV.—The following case is of interest in this connection, although the cerebral condition was complicated by, or caused by, a sunstroke, and the nature of the meningeal effusion may be questioned. At the autopsy chronic ependymitis was found, but the serous effusion was confined to the convexity of the brain. If this effusion can be properly regarded as that of a meningitis and not edema, the case is an example of the serous form of external meningitis.

J. D., age 15, school-boy. Previous history—Boy has always been mentally backward (probably from not having gone to school until 12 years old), but physically well.

Present illness—Had been playing out-of-doors in the sun all day—the day of entrance; in afternoon was sent on an errand; later discovered on a doorstep in an unconscious condition. Admitted to hospital, service of Dr. Jackson. Condition on entrance—Unconscious; head retracted, cyanotic; hot, dry skin; breathing rapid and noisy. Eyes rotated upward and to left; pupils pin-point and stable; conjunctivæ injected; pulse regular, rapid, of poor strength and volume; temperature 110; arms slightly flexed and rigid; legs negative; reflexes present; other organs normal. Treated with ice-cold sponging and packs. Temperature reduced at first; later rose; irregular; fell to normal on the fifth day, then rose and continued between 102 and 103. Profuse vomiting and involuntary ejections. Unconsciousness continued for three days. Temperature required several cold packs each day; nourishment taken; marked rigidity of the neck, trunk and extremities.

Fifth day—Patient seemed conscious, though stupid. Will answer a question by yes or no. Temperature still up. Pulse fairly strong. Three sponge-baths given to-day.

Ninth day—Pupils small, left being somewhat larger than right; neck rigid; constant convulsive movements of extremities; occasional twitching of face; puts tongue out when spoken to; takes a little milk with a spoon; keeps muttering a good deal; constant vigil; passes urine involuntarily; bowels constipated lately. Dr. Lancaster found "no neuritis or other abnormality of fundus."

Thirteenth day—Patient's condition is decidedly worse. There is now profound unconsciousness, with less convulsive movements of hands; respiration rapid; temperature between 101 and 103; pulse weak and rapid; takes very little nourishment; loose movements of bowels to-night after repeated cathartics.

Fourteenth day—Patient failed steadily all day. The

pulse grew weaker and weaker, and the respiration became more shallow, until he died at 11.55 P.M.

Autopsy—Brain: pia extremely distended by a large amount of slightly thickened, cloudy serous fluid. The brain looked as if covered with a thick layer of wine jelly. Vessels of pia markedly injected. The right lateral ventricle had noticeable adhesions between opposite layers of the ependyma; velum interpositum thickened and contracted; no edema of any organs of the body; other organs, with the exception of emphysema of lungs, negative.

Case V.—Under the title of "A Case of Recurrent Basilar Meningitis," Dr. Henry M. Koles reports⁴ a case which, it seems to me, in all probability, was of the serous variety, with effusion into the ventricles. The patient, 23 years old, had previously suffered from several (four) similar attacks of varying, but, as compared with the last attack, less severity. The first followed a boxing bout, when he was thrown against the edge of a table and rendered unconscious. By one attack there was paralysis of one arm and leg. At one period he suffered from attacks of headache, recurring every six months, preceded by vomiting, and confining him in bed for several weeks at a time.

Final attack—Onset sudden; vomiting, headache, restlessness; fever (T. 101), rapid full pulse. Recovery in the course of a few days; return to business followed, after a few hours of the same day, by sudden faintness, dizziness, and nausea; severe vomiting. Again fever, rapid pulse, flushed and anxious face, labored breathing; retraction of the head. During following ten days progressively worse, various paralytic symptoms developing one after another. Symptoms — Pain in head, abdomen and back of neck, and rigidity of muscles of neck. Dilated and stable pupils, exophthalmos, then ptosis and double sixth-nerve paralysis. Neck rigid and immovable. Increased pain in head, and pain down the arm. Restlessness and delirium; maniacal. Left facial paralysis. Highest temperature, 103:

⁴ Med. Rec., Oct. 10, 1896.

pulse, 140. Optic neuritis. Apathetic and delirious by turns. Cheyne-Stokes respiration. Death apparently impending, but instead unexpected gradual improvement and recovery. Treatment anti-syphilitic, though no history of syphilis.

The exophthalmos, double sixth-nerve paralysis, and dilated pupils, are plainly indicative of increased pressure from effusion into the ventricles.

It has already been stated that the chronic cases simulate tumor, and, according to Quincke and Oppenheim, are usually diagnosticated as such. It is highly probable that the "two cases in which signs of the presence of an intracranial tumor were followed by recovery," lately reported by Dr. J. Mitchell Clarke,⁵ were of this nature; and if this interpretation of these interesting cases is correct, they may be quoted here in corroboration of this statement. In both cases there were optic neuritis, intense headache, vomiting of nervous origin and vertigo. In the second case there were, in addition, chills, muscular twitchings and cramps, and pains in the occiput and jaw. Both pupils were dilated, the left being greater than the right, and both reacted sluggishly. There were drowsiness, mental irritability and delirium. Clonic spasms of one hand were also noted. In the first case there were sudden attacks of blindness, lasting two or three minutes. The temperature in both cases was normal or subnormal. The duration of the first case was eight months, of the second, four months. In the first case there were evidently periods of improvement followed by relapses, as the "patient returned to work at irregular intervals." Both cases recovered, but with persisting blindness. The theory of the reporter is that they were cases of tubercular tumor which had become encapsulated, but he does not seem to have taken into consideration the possibility of "serous meningitis," although the common form of meningitis, and other possible conditions, were considered in making the diagnosis.

⁵ British Medical Journal, Feb. 6, 1897.

DISCUSSION.

Dr. Philip Coombs Knapp, of Boston—I suppose that Dr. Prince has spoken, in the part of his paper which he omitted to read, of the importance of lumbar puncture in the diagnosis between this condition and the tubercular form of meningitis. I simply bring this forward now because the last two or three months in Boston have shown very clearly the value of the lumbar puncture in the diagnosis. There has been an epidemic there, some thirty cases having come to the hospital in the last few months, and lumbar puncture has been practiced in every case, with very valuable results, differentiating clearly between the epidemic cerebrospinal form and cases, which presented similar clinical pictures, of tubercular meningitis. The negative findings after lumbar puncture seem to me most valuable in determining between this so-called serous meningitis and the other forms.

Dr. Joseph Collins, of New York—There can scarcely be any doubt that this condition occurs. As to whether or not the process is an inflammatory one depends entirely upon the meaning we put upon the term inflammatory. If we incline toward the view that no condition is inflammatory unless it is attended by a vascular exudate, then the morbid process at the bottom of this condition is not an inflammatory one. If, however, cellular proliferation is an indication of inflammation, we must admit that there are cell changes which go on in the ependyma, *i.e.*, in the internal lining of the ventricles, and, indeed in the pia, that must be considered inflammatory; in short, there is a reproduction of cells, and new cellular formation, and this new cell formation is commensurate with cell disintegration and progresses, the former endeavoring to supersede the latter.

I am glad to hear Dr. Prince say that in all probability there are chronic forms of this condition. I venture to believe that chronic, or subacute, forms may be of more frequent occurrence than the acute. It is possible that some of the obscure intracranial manifestations occurring in children, which we are now unable to interpret, may be due to this morbid state of the ventricles. I am prompted to this remark because of an experience which I have recently had with a case that conformed to the description of hereditary cerebellar ataxia in almost every particular, except that there were *pie'd bot* and peculiar trophic muscular changes. The case was demonstrated before the New York Neurological Society, and the diagnosis of hereditary cerebellar ataxia was confirmed. The patient came to autopsy a number of months ago, and a ventricular distension, very like that described by Dr. Prince in one of his cases, was the only departure from normal that could be found to explain the symptoms from which he had suffered

for many years. I say *explain* these symptoms, but I do not mean to say that the ventricular condition did truly explain the symptoms. At least the cerebellum was apparently normal to the unaided eye, but this does not preclude the existence of changes in the latter which will be discovered later. The case impressed upon me the importance of thoroughly exhausting the possibilities of ventricular conditions before making a diagnosis of structural disease of the substance of the encephalon.

The President—I would like to ask if there was a microscopic examination of the cerebellar cortex in the case to which you refer.

Dr. Collins—No; the microscopic examination has not yet been made.

Dr. Sachs—I think we ought to hold this matter in abeyance; I do not think that Dr. Collins would be willing to say that the ventricular distention which he found in that case had any direct bearing; and until the examination is entirely complete, I do not think it would be wise to have our views distorted by any findings of that sort. I think we must not be too hasty to force a single pathological finding to answer as an explanation for an hereditary disease which has as distinct symptoms as hereditary cerebellar ataxia.

Dr. Morton Prince—Undoubtedly there is a chronic form of this affection, and it is probably just as common as the acute form, but the chronic form generally simulates, and is usually mistaken for tumor. There is present optic neuritis, headache, vomiting, etc.. The principal differential point in the diagnosis is the variation in the intensity and persistence of the individual symptoms. While in tumor the symptoms are fairly constant, in the other condition they may vary largely in their intensity from time to time. The chronic form may be chronic from the beginning, or it may be the outcome of an acute case. The acute cases may get well, or may become chronic cases.

There is also another chronic form which it is claimed exists, at least by Quincke, and that is the neurasthenic form. He claims that in certain cases the symptoms are of a neurasthenic character. This is difficult to prove, because naturally such cases do not come to autopsy; but I would call attention to the second case which I reported, which had been a neurasthenic for a very long period before the final attack, and in this neurasthenic condition she had attacks very similar to the attack which finally ended her life. That case seems to me to be of value as evidence in favor of the statement that the symptoms may be neurasthenic in the chronic form. I am inclined to think this condition may be far more frequent than is generally supposed.

NEW YORK NEURULOGICAL SOCIETY.

Stated Meeting, April 6th, '97. B. Sachs, M.D., President.

THE STATE OF THE REFLEXES IN SUPRA-LUMBAR LESION OF THE CORD.

Dr. Joseph Fraenkel read this paper. He said that it was a general clinical rule that interference with the conduction of the spinal cord leads to an increase in the reflex activity of the centres in the cord below the lesion. The following illustrative cases were cited:

Case 1. A patient of sixty years entered the Montefiore Home fourteen months after the beginning of the disease. During the first six months he suffered from sharp, darting pains in the lower extremities. Extensive bed-sores developed during the last four months of life. The examination showed total motor and sensory paraplegia. There was no tenderness over the spinous process, and no localized atrophy. The autopsy revealed caries of the bodies of the eighth and ninth dorsal vertebræ, with great thickening of the dura at this level, and adhesion to the vertebræ for about three inches. The cord was markedly softened, and the microscope showed extensive changes, mainly of the nature of myelomalacia. Above the lesion there was secondary degeneration of the columns of Goll; below the lesion secondary degeneration of the pyramidal tracts. The process was probably tubercular.

Case 2. A woman, twenty-one years of age, entered the hospital on account of transverse myelitis. The disease had developed without assignable cause, nine months previously, but she had not sought relief until the involvement of the bladder. Six months after the appearance of the first symptom she was completely paraplegic. The examination showed marked anæmia; temperature 102° F.; extensive bed-sore over sacrum; no atrophy; lower extremities decidedly tender and flexed at the hip and knee. There was total motor and sensory paralysis of the lower

extremities. All reflex activity in the lower extremity was lost, yet, on tickling the soles of the feet, they were drawn up. The autopsy showed, at the level of the eighth dorsal vertebræ, a fistula leading into the spinal canal, and a very vascular sarcoma, the size of a cherry, which had pressed upon and softened the tenth and eleventh dorsal segments. The microscope showed that some healthy fibres kept up the continuity of the cord.

Case 3. The patient was forty years of age, and had a good personal and family history. Up to two weeks before admission his disease showed the familiar symptoms of marked pulmonary tuberculosis. At this time the spinal cord became involved in the process. The examination showed the temperature to be 101° F.; there was an extensive tubercular process in each lung; the spinal cord was tender from the lower dorsal region downward; he was greatly emaciated, but there was no localized atrophy. The gait was uncertain, apparently due to imperfect co-ordination. There were numerous areas of anæsthesia and analgesia. All superficial reflexes were present, but somewhat diminished. Fairly good contraction of the quadriceps tendon could be *felt* on tapping the tendon, but it evoked no locomotion. Five days after admission, motor paralysis was found to be more extensive, and the bladder was beginning to be involved. The patellar reflex was much diminished. Shortly after this the patellar reflex disappeared altogether, and a bed-sore developed. The post-mortem showed advanced tuberculosis in both lungs, and a pachymeningitis extending from the seventh to the tenth dorsal segments. The cord was very much softened in this region, and the microscope showed a fair number of healthy fibres. The lumbar cord was normal.

Case 4. This was a boy whose trouble had developed immediately after a severe fall. On admission the extremities presented a peculiar swollen appearance, and pitted slightly on deep pressure. There was a large bed-sore over the sacrum. The temperature was 103°; respirations were abdominal; urine contained considerable albumen; pupils were unequal; abdomen very tympanitic; hyperæsthesia in upper part of trunk, and total anesthesia below. The temperature sense was perverted. The muscles were in a state of complete flaccidity. There was total motor and sensory paralysis. When the soles were tickled the limbs were drawn up. At the autopsy, the

sixth, seventh and eighth dorsal segments were found to be replaced by a fibrous band.

The foregoing cases, Dr. Fraenkel said, showed that occasionally lesions of the cord above the lumbar region did not cause loss of the reflexes. He had found ninety new cases reported in the literature, representing a variety of pathological conditions, distributed in widely different parts of the cord. In the old cases there was absolute loss of motility. When the continuity of the cord is completely interrupted, this can be easily understood, but not so when the interruption is only partial. It seemed reasonable to infer that the motor fibres were always the first ones to suffer in a pathological process. Sensibility was absolutely lost in all cases where section of the cord was complete. The fibres that serve to conduct the deep sensibility seemed to have a more specialized function, and were probably the ones most prone to suffer next to the motor fibres when the cord was subjected to pressure. In nearly every case it was stated that the sphincters were paralyzed. In fifty-one reported cases, and in one of his own, there was total destruction of the continuity of the cord, and in all the lesions were such as to indicate serious injury to the cord. In his opinion Bastian's theory seemed to have met with the most general acceptance. The exact nature of the knee phenomenon was not definitely known. We know positively that the influence exerted by a higher centre could inhibit the reflex, and it seemed reasonable that this superior influence should run in an arc. The centripetal branch of this arc was in some of the ascending columns of the cord to which no definite function had yet been allotted. It should be remembered that loss of the knee-jerk in supra-lumbar lesion was not necessarily a matter of importance in prognosis.

Dr. M. Allen Starr said that he had just been reading a paper covering somewhat the same ground—a paper by Kocher, of Berne. He reported seventy cases of transverse lesion of the cord, and made very positive statements to the effect that loss of the knee-jerk is absolutely diagnostic of total abolition of the continuity of the cord above the lumbar region. Many neurologists had doubted the validity of that proposition, and hence the paper just presented was particularly timely. Bastian's statements had provoked criticism from many quarters. This paper admitted the possibility of a transverse lesion in some cases causing exaggeration, and in other

cases a loss of reflexes. While this might not satisfy our theories, it was an important contribution to the facts.

Dr. Onuf said that Dr. Fraenkel had called attention to the rôle played by the cerebellum in the production of the knee-jerk. We knew further that the pyramidal tracts had an inhibitory influence on the knee-jerk. He desired to advance a theory in this connection. We had not been able to explain many physiological facts except by assuming the influence of so-called inhibition. One important example of this was to be found in the pyramidal tracts, particularly in those fibres passing from the cortex directly to the anterior horn cell. So far as he knew, no one had tried to supply an anatomical basis for inhibition in order to explain the occurrence of excitation in one case, and inhibition in another. Dr. Onuf said that he assumed that when a current from a cell passed from the protoplasmic process towards the nervous process, or from the cell body to the nervous process, excitation of the cell resulted. If, on the contrary, the current passed from the axis cylinder or nervous process towards the cell body, inhibition was the result. These connections were not entirely theoretical—they had been actually demonstrated. He also assumed that the axis cylinder of the pyramidal fibre ended with the terminations around the axis cylinder of the anterior horn cell. The pyramidal fibres, he said, had not only an inhibitory influence on the knee-jerk, but their function was, in general, not a motor one, but an inhibitory one. He would say that voluntary excitation took place through the cerebellum, and that the direct cortical spinal fibres had only an inhibiting influence. A case of absolute paralysis in connection with degeneration of the pyramidal tract had never been reported. By assuming a motor pathway, this inhibition would be found very important in finely graduating the movements. We knew nothing definite about the mode of termination of the fibres around the peripheral neuron. According to the theory just propounded, a complete transverse lesion, interrupting the pyramidal tracts, should cause loss of the knee-jerks. Interruption of the motor cerebellar spinal pathway should cause complete loss of the knee-jerk, for the peripheral stimulus is counteracted by the inhibitory influence of the pyramids. One fact agreed very well with this theory, viz.: the degeneration of cortical spinal fibres (pyramidal tracts) frequently associated with degeneration of the anterior horn cells.

Dr. C. L. Dana said that Dr. Onuf's theory was very ingenious, but it was open to criticism, which, however, should not be made until after further consideration of it. The views of Dr. Fraenkel seemed to him very similar to those of Dr. Onuf, and they were certainly very plausible. The chief problem presented was to explain those cases in which the knee-

jerks were absent, and yet the transverse division of the cord is not complete.

Dr. Fisher asked Dr. Onuf if the pyramidal tracts were no longer to be considered as motor tracts, but rather as simply inhibitory tracts.

Dr. Onuf replied that the pyramidal fibres were not the motor tracts in the sense in which we had hitherto considered them. There must be at least this motor pathway, although there might be another one, through the pons, for example. When we stimulated the cortex, the fibre which gives the motion is the one which passes into the pons and cerebellum and into the cord.

Dr. Fisher asked why, then, with absolute destruction of the cerebellum, as had been done experimentally, we did not get absolute paralysis.

Dr. Onuf replied that *all* of the cerebellum had not been extirpated. He did not claim that only motor pathway was through the cerebellum, but that there must be another motor pathway than the pyramidal tract.

Dr. C. A. Herter said that the cases reported by Dr. Fraenkel were very valuable. His experiences certainly showed that it was possible to have a complete abolition of the patellar reflex with a partial transverse lesion of the cord above the knee-jerk centre. His own impression, gained from clinical observation and from a perusal of the literature, had been that we were justified in regarding a loss of the knee-jerks, in consequence of a transverse lesion high up in the cord, as an evidence that the transverse lesion was a complete one. He was now disposed to think that we had perhaps been rather hasty in considering this a final conclusion. While he had some hesitancy in throwing aside the motor function of the pyramidal tract, he believed the theory presented by Dr. Onuf embodied certain valuable suggestions.

Dr. Joseph Collins said that he had been particularly gratified with the paper, because, when a few months ago he had ventured the statement before the Society that in cases of partial lesion of the cord there was sometimes absence of the knee-jerk, he had been told that this must be an error of observation. But the theory that Dr. Fraenkel had proposed concerning the reflexes presented serious anatomical difficulties. While the theory that the fibre conducts impulses in the direction in which it degenerates was no longer tenable, it seemed to him that, as a general rule, it was true. He thought we must assume some higher influence than the cerebellum to explain the reflexes, and he believed that some function of the somæsthetic area was at work. This was very much in consonance with the theory promulgated by Dr. Onuf. He did not think any one to-day held that the motor tracts were absolutely and only motor. The facts of infantile cerebral palsy

show that defects of the somæsthetic area are not accompanied by paralysis, and it had been shown that the motor impulses starting from the cerebellum were modified in some way.

Dr. Starr queried if it would militate against Onuf's theory if it were considered that the pyramidal tracts were developed in animal life exactly in accordance with the development of the digital extremities. Dr. Spitzka has shown that in the seal, walrus and elephant the pyramidal tract is practically wanting, and develops in accordance with the motility of the digits. This fact was interesting in connection with Onuf's theory.

The president said that in spite of all the facts brought out this evening, the cases under consideration were, after all, exceptional. Granting that in the complete lesion of the cord above the lumbar level the knee-jerks were abolished, there were certainly a number of other cases, which, although the lesion was tolerably complete, the functions were not entirely lost. In Pott's paralysis it was a common thing for the knee-jerks to be exaggerated. He had had an autopsy in a case of dorsal Pott's, in which the cord was completely softened, yet the knee-jerks, instead of being lost, were exaggerated from beginning to end, and there were also contractures. For these reasons it did not seem to him wise to build up theories to explain exceptional cases. We would all be compelled to abandon the theory that the pyramidal tract conducted motion only, or chiefly. There was a fair foundation for the view that there was one cortico-spinal tract, and one cerebellar tract. After an examination of a large number of cases of spastic palsies in both adults and children, he had found very few cases in which there was no paralysis. He felt positive that these cases had more paralysis than spasticity or rigidity.

Dr. Onuf said that the pyramidal tracts had been found absolutely absent throughout the whole cord in a foetus of seven or eight months. This fact had been held to explain those cases of Little's disease in which there was no cerebral disease, and no paralysis, but only rigidity. It also tended to confirm the view that the pyramidal tracts were not strictly motor pathways.

Dr. Fraenkel, in closing, said that he did not doubt that any supra-lumbar disease of the cord would produce spastic paraplegia, but when there is a mechanical and pathological condition interfering progressively with the conducting function of the cord, and sufficient to give motor paralysis, the knee-jerks would be lost. Where there was a total anatomical destruction of the cord, the reflexes were abolished, probably because of the cutting off of the cerebral influence passing through the columns of Gowers. In a few recorded cases there was only partial destruction, yet the knee-jerks were lost as soon as the deep sensibility fibres were destroyed.

NOTES ON SOME ANATOMICAL CHANGES IN THE BRAIN CELLS
IN ACUTE ALCOHOLISM.

Dr. Charles L. Dana read a brief paper with this title, reporting ten cases, and the results of his studies in this direction in the past two years. He had used Nissl's stain for the most part. He stated that what was ordinarily known as acute alcoholic meningitis could not be said to be a meningitis at all, although clinically these persons died with all the symptoms of meningitis. The autopsy would reveal simple congestion and oedema of the brain, and even the microscope would rarely show any migration of leucocytes or anything approaching encephalitis. In some cases not even vascular activity would be observed. Alcoholic meningitis was not primarily a vascular disorder, but a slow poisoning; hence we must study the cell to determine the changes produced. It had been stated by some investigators that all forms of cell degeneration were the same, and that it was impossible, as Nissl claimed, to make out different cell degenerations in accordance with the particular pathological irritant. Whether this were so or not, certainly the microscopical appearances were different in the different cases of alcoholic meningitis, and in other cases associated with delirium and acute disorder prior to death. There was one type of degeneration quite characteristic of those dying from sunstroke, with intense delirium and very high fever. It consisted in a distinct and general pigmentation, involving the larger cells. This sudden development of pigmentary degeneration would seem to be characteristic of acute febrile degeneration, associated with acute toxæmia. Another kind of pigmentary cell degeneration was found in a case of pernicious anæmia. Here the pigmentation involved both the small and large cells. In a case of prolonged use of morphine and whiskey, in which death was due to exhaustion and malnutrition, the brain showed quite a general atrophy of both the nuclei and the cell bodies. There are three types of cell degeneration, viz.: (1) Intense pigmentation of the larger cells, chiefly, with degeneration of the cytoplasm; (2) a general cell atrophy of the body and nucleus; and (3) a good deal of change in the cell body, with many neuroglia nuclei in the peri-cellular spaces. In the cases of alcoholism and alcoholic meningitis it was not possible to make a distinct type of cell degeneration, nor could this hardly

be expected, as these patients die not so much from the alcohol as from auto-toxæmias and from the febrile process.

A CASE OF MENINGITIS SEROSA ACUTA.

Dr. C. A. Herter reported a case apparently coming in this category. The patient was a man, thirty-three years of age, who had enjoyed good health up to a year before his admission. While in the hospital he suddenly developed coarse clonic spasms; the feet were habitually in extension; there was slight clonic spasm in the flexors of the thigh. There was extreme and constant deviation of the head and eyes to the right. The spasms in the arms and legs were fairly symmetrical. The pupils were equal, of moderate size, and reacted fairly to light. The pulse was slow, high tension and fairly regular; the respirations were stertorous. On the following day the right arm was rigid and the left relaxed, and the coarse flexor spasm had disappeared. Then a hemiplegia developed on the left side; the pulse was rapid, feeble and of low tension. Towards the close of the day the patient could be aroused to partial consciousness. On the third day the irregular coarse spasms of the muscles began again, and were most marked at this time on the left side instead of on the right. The right pupil was dilated. On the fourth day the condition was essentially the same, but towards evening the irritative symptoms were less marked. On the fifth day all spasmodic movements ceased, and the patient remained comatose until death occurred. The temperature had been only moderately elevated. The autopsy was made by Dr. Ira Van Gieson. He found over the entire frontal and central lobes a thick layer of fluid between the pia and surface of the brain. The pia was milky, thickened and opaque. In the occipital and temporal regions this membrane was quite normal. The fluid was almost abundant in the frontal region, but was nearly uniformly distributed over the whole centre. The vessels and pia mater of the base were normal. The substance of the brain was pale, and the convolutions only slightly flattened. Microscopical examination showed the vessels of the pia to be largely obliterated. This effusion appeared to have occurred at the time of the onset of the acute symptoms. Probably the hemiplegia was due to pressure on the cortex. A sim-

ilar case had been reported in which the effusion had apparently been due to an infection.

Dr. Dana said that he had seen quite a number of cases of alcoholism presenting an almost identical pathological condition, although such a serous condition of the brain was not the rule in alcoholism. The clinical symptoms, however, did not permit of a differential diagnosis, nor did they indicate a sudden effusion.

Dr. Starr said that at operations on the brain a varying amount of fluid would be found under the pia, irrespective of the lesion present. Slight manipulation of the brain by the surgeon would cause a disappearance of this fluid. For this reason he did not think much stress should be laid upon similar pathological findings.

HEMORRHAGE INTO THE LATERAL VENTRICLES OF A NEWLY BORN INFANT.

Dr. Herter presented a specimen illustrating this condition. The child had been born at six months, and had lived in an incubator for a week. Labor had been difficult, but without forceps. A day or two before death extreme flexion of the toes and right index finger had been noted, together with a general and marked icterus. The autopsy showed nothing abnormal except in the brain. In the latter was a hemorrhage which accurately filled both ventricles.

The President remarked that hemorrhage into the brain had been reported as having occurred at some period of foetal life.

OCCIPITAL NEURALGIA FROM DISEASE OF THE COCCYX.

Oscar S. Brown (Med. Council, Sept., 1896) reports a case of severe occipital neuralgia, of four months' standing, cured by removal of a diseased coccyx. The author found that sudden pressure on the coccyx caused no local discomfort but sharp exacerbations of the neuralgic pain in the occipital region. A hypodermic needle removed a drop of fetid pus, and operation revealed necrosis of the entire coccyx, and about a dram of offensive pus. The operation was done several years ago, and the patient has not had "the slightest twinge of pain" since.

PATRICK (Chicago).

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 22d, '97. President, Dr. Charles W. Burr, in the chair.

Dr. A. A. Eshner exhibited a patient with
BASILAR MENINGITIS. (See this journal for March, p. 167.)

D. Chas. K. Mills.—The case, I think, is rather difficult for diagnosis. I saw this man a year or so ago. From the symptoms then presented the diagnosis of a form of chronic degeneration of the nervous system seemed to be justified. I reported the case as probably one of posterior sclerosis of aberrant type. The man has no headache. He has loss of hearing, which is gradually progressing, and which we sometimes see in cases of chronic degeneration of the nervous system. It is not impossible that this is one of those cases of chronic degenerative disease which will finally end as a case of paralysis of the insane.

Dr. Charles K. Mills reported five cases of

HEREDITARY TREMOR,

and exhibited one of the patients.

Case 1. Mrs. E. P., forty years of age, presented herself at the neurological service of the Woman's Medical College. The patient has had tremor in both upper limbs, greater in the left, since her earliest childhood. The tremor is increased by excitement or by grasping an object, though when she is in repose there is scarcely any tremor in her hands. The tremor in the head has only been noticed for eight or nine years, and is attributed to a fright from a man supposed to be insane. This tremor in the head is greater than in the hands, and is of a slightly rotary character. The patient has never noticed any tremor in the lower limbs. She is very excitable. The knee-jerks are present but not notably exaggerated. There is no ankle-clonus. The sensation for touch and pain is normal in the entire body. There is no inguinal tenderness. Inframammary tenderness is found on both sides; is a little

more intense on the left, but the whole area of the stomach is tender to pressure, and the patient suffers from indigestion. There is no tenderness along the spine, and no limitation of the visual fields. No history of the parents, and no statements in regard to tremor in the father's family, can be obtained. The mother's family is not known.

The daughter has had a similar but more intense tremor, noticeable only in her hands, ever since birth. Her head is not affected. She is not as excitable as her mother. She has a tremor at all times, even when not excited. She has a child of four years, who has no tremor.

Case 2. T. J. E., forty-five years of age, a resident of Mechanicsburg, Pa., was seen with Dr. J. H. Boyer.

Since about 1882 he has had a peculiar jerking and tremulous movement of the head, which has remained almost unaltered. This tremor has a tendency to draw the head backward and to the right. The chin and whole head turn to the right. Some tremor of both hands and arms is also observed. Any occupation increases the tremor of the head. He occasionally takes a drink of whiskey to steady himself. He has no nystagmus, and the special senses apparently are not affected; the knee-jerk is a little plus.

The father of the patient, now eighty-five years of age, has a similar tremor of the head, which developed when he was sixty-five. A sister, of forty-two years, is similarly affected, and in her case the tremor was first noticed at the age of thirty-two or thirty-three.

Dr. A. A. Eshner exhibited a case of

CONGENITAL TREMOR.

He stated that persons occasionally present persistent tremor without obvious cause or other morbid signs. Sometimes the shaking may be observed in several members of the same family, and there may be an evident transmission from parent to children. The tremulousness may not appear until late in life or it may develop early. The earlier the manifestation, the stronger would be the reason for suspecting a structural or organic basis. On the other hand, slight tremor in an infant might readily be obscured by the natural incoordination of movement present at this time of life. Apart from the rotary and nodding movements of the head, not rarely seen in rachitic

children, tremor is not often observed in infants in the absence of structural or organic disease.

The case which he reported was designated as one of congenital tremor on account of the early period in life at which the movements were noted—with the possibility of its still earlier presence; of the absence of evidence of an organic cause, and of the freedom of other members of the family from a similar disorder.

M. F. M., now thirty-six years old, presented himself in the autumn of 1893, at the Infirmary for Nervous Diseases in the clinical service of Dr. S. Weir Mitchell, complaining of a tremulousness that had been present as long as he could remember. His recollection carried him back to boyhood, when the right hand shook distinctly in school work. He was unable to state with certainty whether or not both hands were tremulous, but he thought they were. No tremor had been noticed by his parents during infancy. For perhaps fifteen years he had noticed a certain weakness of the legs, which would at times give way in walking, and staggering result. This was particularly marked in ascending acclivities. Such muscular efforts were unattended with tremor, although for a period of eight or ten years, when one leg was thrown over the other, a shaking—possibly clonus, but more probably simple tremor—would be set up in the foot.

The symptoms noted gradually progressed in intensity. For a moderate period of the time the patient, in the pursuit of his occupation as a laborer in a mill, had been obliged occasionally to carry heavy loads, and it was noticed that the tremor was increased after such efforts. The tremor was greatly aggravated by observation, by unusual surroundings, by emotions, by excitement, by laborious work, and by the chewing and smoking of tobacco. It was absent during sleep, and as a rule during rest, and was essentially an intention tremor. The drinking of beer would for a short time be followed by a lessening of the movement, with, however, subsequent aggravation. The trembling was worse when constipation existed, and also when diarrhea was present. The patient related that, as a child eight or nine years of age, he would from time to time be frightened by the thought of a strange person in his bedroom, and he would not be quieted except by the presence of his father, although he slept with

an older brother. In disposition he was irritable and easily angered.

In standing, the patient appeared quite steady, although he felt a slight shaking of the right hand. The movement was quite beyond his control, and was increased by efforts to suppress it. When the arms were extended at right angles with the trunk, the fingers and hands were set into rhythmic tremor, as if from alternate action of the flexors and extensors of the carpus. Muscular activity was soon followed by fatigue, while support afforded subjective relief. On attempting to touch the tip of the nose or of the ear with the fingers, and on attempting to bring the fingers of opposite hands together, coarse oscillatory movements took place, rendering variable and uncertain the result, which was sometimes successful, sometimes not. The knee-jerks were active, full and strong, rather exaggerated than otherwise. Dorsal flexion of the foot on the leg elicited only an abortive attempt at ankle-clonus. There was no derangement of sensibility, common, painful or thermal. The pupils were equal, regular, and reactive to light. Nystagmus was not present. Speech was not interfered with. There was no evidence of disease of heart or lungs. The appetite was good. The tongue was coated slightly, and protruded in the median line; it shared in the general tremor, particularly at its tip. Costiveness was usually attended with nausea, acidity and vomiting. Hearing was impaired upon the right side. Memory for recent events was enfeebled, that for remote events preserved.

The patient had suffered from enteric fever at the age of twenty-five years. He denied sexual excesses, and had no knowledge of venereal infection. During a period of twelve years, until within a short time of coming under observation, the patient had indulged periodically in alcoholic excess. He had used tobacco freely, but had taken tea and coffee in moderation. There was no history of blow, nor injury, nor traumatism of any kind. The father of the patient had been killed in a railroad accident. The mother was living at the age of sixty-five or sixty-six years; she was believed to be rheumatic, and suffered from time to time from attacks of precordial pain, which were relieved by the ingestion of a glass of water. One brother was living and well; two brothers were dead of enteric fever—one of these suffered from stuttering speech.

The patient's handwriting showed the pronounced tremulousness, while that of the brother showed nothing abnormal. Tracings of the patient's tremor were noteworthy for their want of uniformity and of regularity, although the rate of movement appeared to have been about six per second. The movement in the two hands was approximately, if not absolutely, synchronous.

It is not impossible that there exists in this case a degenerative or sclerotic process, necessarily slight in degree, involving the lateral columns of the spinal cord, although the symptoms are scarcely well enough defined to render such a conclusion at all certain. As opposed to this diagnosis must be considered the absence of ankle-clonus and of other spastic phenomena, and of noteworthy muscular disability, as well as the early onset and the slow progress of the symptoms. Multiple sclerosis may be excluded from the absence of nystagmus and of peculiarity of speech.

Dr. Chas. K. Mills exhibited a case of

CEREBRAL DIPLEGIA.

J. M. C., a white girl of eighteen years, was born with instruments after a very hard labor, and for some time succeeding birth was supposed to be dead. She first walked when she was fifteen months old, and it was noticed that she dragged the right foot, and that she waddled when she ran. When she was eight years old she had "scarlet fever and measles at the same time," which was followed by a partial loss of voice. A slight trembling of the limbs was noticed before the attack of scarlet fever, in fact, the mother of the patient cannot state just when the tremor was first observed. There is no history of a similar condition in any branch of the family, and the other children are well. The patient has had no convulsions, and has made fair progress in school.

Present condition—She is a well-nourished, intelligent young woman. The tremor is most marked in the head and right arm. The movements are coarse and jerky in character; those of the head being lateral, anteroposterior and half-rotary. The head, trunk and extended arm seem to move together. The tremor of the right upper extremity involves the whole arm, and is partly lateral and partly

rotary, and is most marked in the hand. The left upper extremity is affected with tremor of the same character, but less in degree. The left lower extremity is more affected than the right with the same coarse tremor, lateral and rotary in direction, and more marked distally. The knee-jerks are plus on both sides. Ankle-clonus is found on both sides, and is especially marked on the right, where it is of the persisting variety. The right foot is dragged slightly when the patient walks. The hands are cold and dusky; the feet also are usually cold. The right leg is about one-half inch shorter than the left, and smaller throughout.

Dr. A. A. Eshner.—I have had the opportunity of seeing the girl shown by Dr. Mills, at the clinic of Dr. S. Weir Mitchell. The case was looked upon as one of cerebral birth traumatism. To exclude the possibility of chorea, the girl was treated for a month by the hypodermic use of Fowler's solution in ascending doses, and under this treatment she did not make the slightest improvement.

Dr. Wm. G. Spiller.—The history of difficult labor, the dragging of one foot in walking, the retardation of growth, the bilateral ankle-clonus, etc., are in favor of the diagnosis of diplegia with greater involvement of one side. The tremor has nothing of the character of chorea, and we may probably dismiss all thought of this disease. It seems to have become much more evident after the attack of scarlet fever, when the child was eight years old. After acute affections it is not uncommon to see the development of organic nervous disease. It is, therefore, not surprising that tremor should develop in this case after measles or scarlet fever. I am inclined to think that the tremor is due to a lesion at birth, especially as I have studied a case in which a cortical sclerosis was produced probably in the first years of extrauterine life and was followed by the development of epileptic convulsions at the age of ten. A similar case of infantile paralysis with development of epileptic attacks about this same age, has been reported by v. Monakow. I am inclined to think that the tremor in Dr. Mills' patient is a part of the diplegia, and that the infectious disease at the age of eight was only the indirect cause of its development; in other words, that it was an "*agent provocateur*."

Dr. A. G. Thomson read a paper on

ISOLATED, TRAUMATIC, BILATERAL PARALYSIS OF THE ABDUCENS NERVE.

A man of forty-three years, with no history of specific

or spinal disease, a year and a half ago fell a distance of thirty-three feet, from a scaffolding to the ground. He regained consciousness after an hour and a half, and found that he had complete bilateral paralysis of the sixth nerve, and that the right side of the body was bruised. There were no signs of fracture of the skull, nor of injury of any other cranial nerves. He recovered from his bodily injuries within a few days, and was able to return to his work. The bilateral ocular paralysis is still complete. Dr. Thomson thought the lesion was caused either by a bruise or tear of the nerves in their course from the pons to the sphenoidal fissure.

Dr. F. S. Pearce.—I have seen reported a case of bilateral paresis which the author claimed was probably due to a dystrophy of the external recti muscles. The condition was congenital. Dr. Thomson's explanation is quite plausible.

Dr. Wm. G. Spiller.—Cases of sudden paralysis of the sixth nerve from unknown causes have been reported, but trauma seems to have produced the paralysis in the patient described by Dr. Thomson. I agree with him that the lesion was probably not nuclear. It is possible that a small hemorrhage might destroy both sixth nuclei, but it is difficult to understand how the seventh nerves could entirely escape in such a lesion. The knee of the seventh nerve embraces the nucleus of the abducens very closely, and for a long time was thought to receive fibres from it. I am more disposed to think the lesion is probably at the point of exit of the sixth nerves from the bulbopontine junction, at which place they are close together.

Dr. Joseph Leidy read a paper entitled

NEURALGIA OF THE CRANIAL NERVES AS A SYMPTOM OF
MALARIA.

He stated that occipital neuralgia has frequently been observed as a symptom of uremia, especially the uremia resulting from the atrophic form of chronic diffuse nephritis.

The first case was one of arteriocapillary fibrosis. During the first year there were gastrointestinal disturbances; during the second year, neuralgia, principally of the occipital, inferior dental and sciatic nerves, and evidences of renal involvement, albumin and low specific gravity of the urine; during the third year involvement of the cranial nerves with evidences of cerebral affection, *i. e.*, paresis

rather than paralysis, of the tongue and vocal cords, and periodic attacks of dysphagia and dysphasia.

Finally the cardiac action became insufficient. The respiration during eight days was of Cheyne-Stokes character, but the patient rallied and was able to go about without assistance, and to attend to business. The Cheyne-Stokes respiration was noticed at intervals afterward, and the patient finally succumbed to his disease.

Certain of the symptoms were attributed to the contracted kidneys; others to interference with the cerebral circulation at the base of the brain. The speaker considered the temporary recovery after the attack of Cheyne-Stokes respiration of interest, and referred to a similar case in which the recovery was permanent. He spoke especially of the slowly developing nephritis as a part of a general arterial degeneration without changes, for a time, in the renal excretion, and of the dysphasia due to paresis and incoordination of the movements of the tongue.

The second case well illustrates the importance of microscopic examination of the blood in cases of sudden loss of consciousness.

The patient was admitted to the hospital in an unconscious condition. An examination showed albumin and high specific gravity of the urine. The suspicion of uremia was aroused. The microscopic examination of the blood revealed the plasmodium *malariae* in great abundance. There were ameboid and pigmented forms and several flagellated bodies. The patient was soon seized with severe rigors. In addition to treatment directed to the kidneys, quinine in ten-grain doses was given every hour until a drachm had been administered. Improvement was observed within twenty-four hours; consciousness returned, and the patient made an uninterrupted recovery.

The history was as follows: The man had left the swamps of North Carolina to place himself under treatment in Philadelphia for obstinate neuralgia and vertigo. He grew rapidly worse while on the vessel, and after arriving in this city fell unconscious in the street while on his way to the hospital. The speaker regarded the case as one of malarial nephritis.

Dr. James Tyson.—The frequent association of neuralgia with Bright's disease, especially neuralgia of the posterior auricular nerves, is a recognized condition; at the same time

the independent occurrence of neuralgia due to other causes is so frequent that I should hardly consider this symptom of diagnostic value.

The first case, it seems to me, is admirably explained by supposing that there was a primary general arteriosclerosis in which the vessels of the kidneys shared ultimately with the other blood vessels, and that this sclerosis was responsible for the lesion in the internal capsule and also for the renal symptoms which came on as a later manifestation of the disease. It is often a nice question for diagnosis whether the renal disease is primary and the vascular disease secondary, or whether the vascular disease is primary, involving more or less the blood vessels of the system and the kidneys. I think that this latter condition is found to produce other symptoms prior to the renal symptoms, as was the case in Dr. Leidy's patient.

CONCERNING THE HISTOLOGICAL CONDITION OF THE PERIPHERAL NERVES AND THEIR CENTRES AFTER SECTION. Wiener klinische Wochenschrift, April 29th, 1897. By Arthur Biedl.

With the exception of the first paper published by Nissl in 1890, we have no detailed statements in regard to the changes which occur in the cell body after section of the nerve fibre (Biedl). Biedl has operated on three dogs and three rabbits by removing a portion of the sciatic nerve in the upper part of the thigh. The animals were killed at different periods after the operation, and their spinal cords and sciatic nerves were examined. The results of his investigations are as follows:

Degenerative changes may be found three days after section of the nerve in the cells from which these fibres arise. These changes consist in rarefaction and coarse, granular disintegration of the chromatic substance. At the same time degenerative changes may be observed in the peripheral end of the nerve, while the central end except at the point of section, remains normal. Two days later the granular disintegration of the cell body is more distinct, and fine granules may be seen. The first traces of degeneration may be noticed in the intramedullary portion of the anterior roots. Eight or nine days after the section the ganglion cells present the appearance of the fine, granular, so-called molecular disintegration. The destruction of the cell obtains its greatest degree about the eighteenth day, and consists of fragmentation of the cell body and of transformation of the fine, granular into homogeneous degeneration. The degeneration of the peripheral end of the cut nerve at this time is no longer active, but nearly the whole central end presents the appearances of beginning degeneration. After twenty-eight days the cells are more deeply stained and are homogeneous, but with indications of the formation of cellular processes and nuclei. These are probably the first manifestations of the process of regeneration.

SPILLER.

Periscope.

With the Assistance of the Following Collaborators:

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ANATOMY AND PHYSIOLOGY.

SENSITIVE NERVE ENDINGS IN STRIATED MUSCLES.

Dr. C. Rouget (*La France Méd.*, July 24th, 1896) studying the motor nerve endings in tadpoles by means of impregnating living muscles with methylin-blue, was able to determine the existence of sensitive muscular nerves which terminate on the striated fibres, but outside of the sarcolemma, while the terminal motor nerves are in direct contact with the contractile substance. These two classes of nerve endings are, therefore, not situated on the same plane in the fibre, but are separated by a narrow space, which allows of their being photographed (microscopically) simultaneously. The sensitive endings never present the sudden swellings or inflections like the axis cylinders of motor nerves, but always retain their characteristic mode of ending, such as, for instance, in the cornea, the skin, etc. Furthermore, the course of motor nerve endings, always running parallel with the axis of the striated fibres, differentially from sensitive nerves, the endings of these being directed obliquely or transversely to the muscular elements. Again, each striated fibre is supplied with one or more motor nerve endings, while those of the sensitive fibres are far less numerous, with quite long intervals between them. These nerve endings are most thickly strewn in the deeper layers of muscles, where the terminal motor fibres are also most abundant. The author concludes that the two foregoing classes of nerves, being in direct contact with the contractile elements, form, with the muscular fibre, the hypothetic nervous arc of Charles Bell, by means of which a cerebral impulse is transmitted to a muscle by one nerve, and the condition of the muscle is communicated to the brain by another.

MACALESTER.

ON THE COURSE OF THE TASTE FIBRES.

In the *Edinburgh Medical Journal*, for April, 1897, A. F. Dixon examines the arguments in favor of the passage of the taste fibres to the brain by the fifth nerve roots, and those which opposed to this tend to show that they pass in with the roots of the facial and the glossopharyngeal. On anatomical grounds, and from the results of embryological experiments by himself and others, he is inclined to favor the latter view of the case.

ALLEN.

THE INTERNAL SECRETIONS OF THE THYROID AND SUPRARENAL GLANDS. W. D. Halliburton, M.D., F.R.S. (*The Practitioner*, Jan., 1897.)

Recent research, according to the above authority, has shown that most, if not all, of the so-called ductless glands form a secretion, and

this "internal secretion" leaves the gland by the venous blood and the lymph, to become distributed to other parts of the body. Many glands which possess ducts and form an "external" secretion, also form an "internal" secretion, like the liver, pancreas, testis and kidney. Among ductless glands the thyroid has attracted most attention, and practical clinical results have closely followed experimental research, notably in the treatment of cretinism and myxœdema. The suprarenal bodies have recently also excited much interest, but as yet no sure results have followed practical use of suprarenal extracts in the treatment of Addison's disease. Halliburton turns his attention especially to the physiological facts concerning the thyroid and suprarenal glands, which he claims lie at the roots of pathology and form the groundwork for the rational treatment of disease. When ductless glands were first studied from a scientific standpoint, they were supposed to be excretory organs rather than secretory. So the thyroid was supposed to excrete a mucinoid substance, and when the gland was absent mucin was supposed to accumulate in the body generally, whence came the diseases myxœdema, sporadic cretinism and cachexia strumipriva. Halliburton claims there are two erroneous suppositions in this hypothesis: first, the colloid material poured into the acini by the thyroid epithelium was believed to be mucin, but it is not; and second, mucin does not collect in the body generally. Mucin is only temporarily superabundant, as in all imperfectly fibrillated and embryonic connective tissues. When these tissues become permeated by fibres and by fat, the mucin disappears. The most marked symptoms of myxœdema are of a nervous character, both in human beings and in animals which have undergone extirpation of the thyroid. The colloid is now considered a secretion. It has been traced into the lymphatics, and the part to which it is especially of service is the central nervous system. When the "internal" secretion ceases the nutrition of the brain is particularly affected. Since it became practically known that thyroid extracts relieve myxœdematous and similar conditions investigators have sought for the active chemical agent or agents secreted by the thyroid, and to which the extracts owe their efficacy. Though this search is not yet complete some interesting facts have come to light. Among these is the discovery that there are certain substances in the thyroid which contain iodine in organic combination. Bauman, of Freiburg, found this *thyroidin*, an element hitherto unknown as an integral part of living animal structures. It is claimed and some clinical evidence seems to prove, that this thyroidin exerts beneficial effects just as do the thyroid extracts. It has also been found in small quantities in the thymus. Thyroidin is a proteid found in the colloid substance, which contains both iodine and phosphorus. But whether the proteids of the thyroid alone contain the active principles or whether the non-proteid constituents, too, are of influence, as Fränkel and Drechsel claim, is yet to be determined. The thyreo-antitoxin found by Fränkel has as yet not been proven efficacious by clinical trial.

Then turning his attention to the supra-renal bodies, Halliburton simply and concisely reviews the work of various experimenters, notably Vulpian, Manasse and Fränkel, concluding that the sphymogenin of Vulpian is the active agent in the production of heightened blood-pressure. Sphymogenin is a reducing substance of unknown chemical nature, but certainly contains nitrogen, and acts in some regards like pyrocatechin, but differs from the latter in that it does not reduce Fehling's solution. Fränkel claims sphymogenin is a nitrogenous derivative of the ortho-dioxy-benzene series, and has promised further experiments relating to its practical use.

STERNE, (Indianapolis).

THE INFLUENCE OF INTELLECTUAL WORK ON THE BLOOD-PRESSURE IN MAN. By MM. A. Binet and N. Vasschide. (Paris, Psychological Review, January, 1897.)

Mosso's sphygmomanometer was used. The principle of its application is as follows: Two fingers of each hand are placed in rubber finger-tubes, and through these tubes are exposed to the pressure of water; this pressure is varied by means of a piston and measured by a mercury manometer, which registers at the same time the pulsations of the fingers. If we begin with 0 and increase gradually, we find a regular change in the amplitude of the pulsation; this is very small at first, grows and reaches a maximum, decreases again and finally disappears; thus the amplitude does not vary directly or inversely with the pressure; there is a most favorable degree of pressure, equal on the average to 80 mm. of mercury at which the pulse attains its maximum amplitude.

The mental effort tested was a series of easy calculations—two figures multiplied by two figures—and lasted about two or three minutes. The subject closed his eyes, knit his brow, leaned his head a little forward. Ten experiments in all were made on different days, but on the same hour and under strictly comparable conditions. It was found that a pressure of from 100 to 120 completely suppressed the pulsation during repose and intellectual labor. As soon as the mental calculation begins there is an increase in the pulsation. The first three or four pulsations which register themselves are usually of same character as the preceding; sometimes they are slightly shortened—an effect probably due to the vascular constriction which was habitual to the subject at the beginning of intellectual work. Then the pulsation increases; it doubles in size or becomes twice and often three times as great. This increase in amplitude maintains itself, in general without increase or diminution, and with great regularity during the whole of the mental calculation; when the subject has found the solution and has given it, there is no sudden diminution of pulsation; it may retain its amplitude without change for fifteen seconds, sometimes even longer; then the pulsation begins to diminish very gradually; finally it recovers the same amplitude that it had at the beginning of the mental calculation. This return to the original condition shows that the change is not due to the apparatus but to the physiological condition of the subject.

Three subjects in all were tested with much the same result.

CHRISTISON.

CLINICAL NEUROLOGY.

MIND-BLINDNESS—FOR OBJECTS ONLY. (Lépine, Lyon Medical, May 16, 1897.)

The author first defines the word blindness as an inability to understand the meaning of the words read, although the subject may read fluently. He then reports the case of a man aged thirty, well educated and intelligent, who was supposed to be in the first stages of general paralysis of the insane. He was, however, sufficiently intelligent, understood all that was said to him, wrote perfectly, spoke fluently, and had apparently no trace of aphasia. Central vision was somewhat impaired, and the visual fields somewhat restricted on the left side. His writing was perfectly correct, but he could make a drawing of nothing, even copying of the most simple objects being impossible; neither could he recognize persons or objects except those with whom he had been very familiar.

The case is not unique, although similar reports are exceedingly rare, and the author refers to only one source of bibliography—a paper by Mueller in the Arch. für Psychiatrie, 1893. PATRICK.

HYSTERICAL APPENDICITIS AND PERITONITIS. (Medical Week, Paris, April 2d, 1897.)

At two meetings of the Société Médicale des Hospitaux, Dr. Rendu called attention to the above condition. Dr. Talamon (Medical Week, April 2d, 1897) discusses the difficulties entering into the diagnosis of appendicitis complicated by the presence of hysteria. In one of Dr. Talamon's cases operation was performed; recurrent appendicitis was present.

Dr. Talamon suggests that two kinds of cases are to be distinguished. First, those in which hysteria is the only cause of the condition, there being no lesion of the appendix—hysterical pseudo-peritonitis. Secondly, cases in which with mild appendicitis the symptoms are exaggerated by hysteria so as to suggest the presence of perforative appendicitis and diffuse peritonitis.

In the same connection Prof. Hayem reports a case of incoercible vomiting lasting for more than a year, and finally developing into symptoms indicating the presence of appendicitis. An operation for appendicitis was performed. The appendix was found enlarged and adherent, but no trace of pus was visible.

This case is in favor of Dr. Rendu's contention that vomiting in hysterical subjects should not always be considered as of purely nervous origin; in some cases it may have an appendicular cause.

MITCHELL.

RHEUMATISM AND CHOREA

Churton (Brit. Med. Jour., Sep. 19th, 1896), from an examination of 552 cases of rheumatic fever, and 157 cases of chorea, together with a study of the current literature on the subject, deduced the following conclusions: (1) The postulated toxin (x) being accepted as an essential element in the causation of rheumatism, depressing conditions (y) determine the first position or locus of the disorder—that is, what cells or tissue (z) the toxin shall strike. (There is probably a quite separate and independent causation for (x). In 91 cases of rheumatism in which the incidence of the chill, strain, etc., was recorded with precision, and also the joints or parts first affected, it was seen in every case—or with very few, and doubtful, exceptions—that the part receiving the impact of the conditioning cause was the first to become disordered by the toxin—for example, wetting of feet always caused arthritis first in the lower extremities; of shoulders, in the upper extremities. (2) If (x) and (y) are given, the position of (z) can be stated within certain limits. (3) If (y) is a fright, shock, or intense excitement, (z) will be the nervous system; in the developing brain of a child the result is usually chorea; in adults it may be delirium or coma, perhaps hyperpyrexia. (4) Arthritis or endocarditis may follow the nervous disorder, since the symptoms themselves may become causes of depression of the tissues, and thence of multiplication of micro-organisms and toxins. (5) Similarly chorea may follow arthritis; but (6) arthritis is never the first result of fright; and (7) chorea is never the first result of chill, unless fear or brain excitement accompanies the chill. (8) A man who, being rheumatic, and having no other known disease, is accidentally subjected to strong excitement—a quarrel—and in a few hours develops chorea, is an "experiment devised by nature" to prove that the essential cause of the two disorders is the same, and that only the conditioning (localizing) causes are different. In non-rheumatic persons, even young children, brain disturbance does not cause chorea.

MITCHELL.

NEUROPATHOLOGY.

POSTEPILEPTIC ASYSTOLE. (*Revue Neurologique*, No. 6, 1897.)
By C. Féré.

The coincidence of cardiac affections with epilepsy has been noticed, and the latter has been attributed to circulatory disturbances; sometimes to cerebral congestion in mitral disease, and sometimes to anæmia in aortic. Lesions of the myocardium may have the same results. On the other hand, epilepsy may cause cardiac affections. The increase in the arterial pressure during the epileptic attack seems to be due to more energetic contraction of the heart; to contraction of the small vessels, and to contraction of the muscles, which hinders the flow of blood from the capillaries into the veins. That the epileptic attack is accompanied by a considerable increase in the arterial pressure may be regarded as a demonstrated fact. The fall of the pressure after the attack may be explained by the lessened tonicity of the muscles of the limbs, by the paralytic dilatation of the peripheral vessels, and by the diminished action of the heart. The cardiac affection may explain the cases of death following a single convulsive attack. This may be due to syncope from arrested cardiac action, or to cardiac rupture.

Féré reports a case in which the first attack of epilepsy occurred at the age of forty-two. After a series of epileptic convulsions, an attack of acute asystole developed, with the signs of imperfect cardiac action, such as: cyanosis, swelling of the veins of the neck, arrhythmic, feeble condition of the pulse, etc. There is no morbid manifestation in the circulatory system of this patient at present. SPILLER.

PARADOXICAL PUPILLARY REACTION.

In a very interesting article, Dr. H. Frenkel (*Gazette Hebdomadaire Médecine*, August 2d, 1896) describes very carefully an ophthalmological and neurological study of a patient with the above rare symptom. The man presented himself for the treatment of an intermittant alternating divergent strabismus. Apart from a slight posterior staphyloma, the fundus was normal. The visual field on the right side was slightly contracted with a normal succession of color; on the left side there was a very marked contraction for white and for all the colors, on the nasal side, in fact, a left nasal hemianopsia. The left eye had, too, a slight paresis of the internal rectus muscle, and a small degree of ptosis. The left pupil was much smaller than the right when at rest. Pupillary reaction was normal for accommodation, but upon throwing the light upon the left eye, the pupil would remain motionless for five or ten seconds, then dilate slowly to double or more than double its previous diameter, remaining dilated until the light was withdrawn, when it would rapidly contract again. The same phenomenon was less pronounced upon the right side. As the patient was found to have an aortic insufficiency, with enlargement of the heart, it might readily have been concluded that the cause of the paradoxical reaction was in the encephalon, due to localized hemorrhage or other results of the valvular disorder; but Dr. Frenkel, upon closer examination, drew a different conclusion. It was found that upon prolonged examination of the eye, the patient, while accommodating for distant vision allowed his right eye to deviate during the examination of the left; the left pupil consequently would at first remain stationary, then, as the right eye abducted, would dilate, thus giving the supposed paradoxical reaction. A contrary test was also instructive: the pupils were unchanged while the patient accommodated for a near object, so long as the eyes did not move; as soon as the internal recti began to grow fatigued, but not till then, dilatation

began. Among Dr. Frenkel's other conclusions, he states that he does not believe the paradoxical reaction is in any way an abnormality; that the dominant condition which makes it possible to observe a pupillary dilation *during* lighting, but not *in consequence of it*, is the presence of Argyll-Robertson's symptom, although there has been one case of paradoxical reaction recorded in a hysterical patient, in which this sign was wanting.

MITCHELL.

THE ARGYLL-ROBERTSON PUPIL.

In a clinical study published in the American Journal of the Medical Sciences, for July, 1896, T. K. Monro reviews the different theories proposed to explain this condition. That the lesion is situated in the efferent portion of the reflex arc running from the optic nerve to oculo-motor nucleus in the motor fibres of the cervical cord, or in a cilio-spinal centre in the cord or medulla, the writer considers equally untenable. His own conclusions are that reflex in diplegia is probably due to a degeneration in the nucleus of origin of the third nerve.

SHIVELY.

NERVOUS TROUBLES OF HEPATIC ORIGIN.

Dr. Leopold Levi (Journal de Méd. et de Chir. prat., July 10th, 1896) demonstrates the important rôle played by the liver in the pathogenesis of certain nervous disorders, which, compared with those of nephritic origin, might justly be called nervous hepato-toxæmia. The graver manifestations are: Hepatic coma, icteric coma, hepatic delirium, folly, convulsions, etc. The minor disturbances are: (1) Changes of character during the course of the disease, with or without icterus being present. The patients lose their sense of humor, become despondent, gloomy and sensitive, taking no more interest in their surroundings, and preferring to live retired without communicating with people. At times they are exacting and quarrelsome, and often develop hypochondriacal and suicidal ideas. (2) Sense of fatigue (muscular asthenia), that often resembles neurasthenia, the patient exerting himself to the utmost in endeavoring to do light work. (3) Cephalalgia, vertigo, disorders of sleep (insomnia or somnolence), and hepatic drowsiness, are common manifestations of hepatic toxæmia. (4) General disturbances of sensibility, pruritus, vasomotor troubles and toxic nephritis, were also observed. (5) Changes of reflexes, the knee-jerk being absent in some and exaggerated in other cases. (6) Ocular troubles (hepatic ophthalmia), comprising xanthopsia, hemeralopia, retinitis pigmentosa, ambliopia, transitory pupillary oedema and affections of the pupils. The author, experimenting on dogs, produced artificial lesions of the liver, and observed subsequently that their character changed entirely, they becoming quite untractable.

MACALESTER.

MENIERE'S DISEASE.

Lemarié (Annales de Maladies de l'Oreille du Larynx du Nez, et du Pharynx, 1896, Med. Record, August 8th, 1896) reports good results from the use of pilocarpine in the case of a man of thirty-five, who suffered most severely from attacks of aural vertigo. They developed suddenly in the course of chronic otitis media, involving both ears. After a week's treatment with various remedies he was given hypodermic injections of pilocarpine once a day, the patient remaining in bed for about two hours after the administration. The dose was at first 4 milligrams of a 1 per cent. solution, which was increased by one milligram every two days. Fifteen days after the beginning of the treatment he was able to go about the hospital, and in two weeks more was practically cured.

PATRICK (Chicago).

THE
Journal
OF
Nervous and Mental Disease

AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-third Annual Meeting, held at St. John's Parish Hall,
Washington, D.C., May 4th, 5th, and 6th, 1897.*

The President, Dr. M. A. Starr, in the chair.

AUDITORY APHASIA.

By HOWELL T. PERSHING, M.D.,

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The term auditory aphasia, used to designate the sum total of defects in the use of language caused by a lesion of the auditory centre for words, or of the fibres immediately connected with it, is to be preferred to the other names used for the same group of symptoms. It is more definite than sensory aphasia, which includes the symptoms caused by injury of either the auditory or visual centre, or of both; and on the other hand, it is far more comprehensive than the term word-deafness, which applies to only one, although the most important one, of a number of related symptoms.

The following case is typical of the clinical aspect of auditory aphasia:

J. W. Long, aged 70, was very healthy during middle life, but after a severe attack of typhoid fever, in 1882, he suffered a great deal from chronic bronchitis and asthma. During the last week of March, 1896, on account of dyspnea, he passed most of the time at night in a chair, and his feet and ankles were somewhat swollen. On the morn-

ing of March 31st he had risen from his chair and was putting on his shoes, when he suddenly fell, unconscious. The physician who saw him an hour later found him still unconscious, with the right face, arm and leg paralyzed. The pulse was rapid and weak, but responded well to alcohol and digitalis, under the influence of which consciousness rapidly returned and the swelling of the feet disappeared, but then it was found that the patient could neither speak intelligibly nor understand what was said to him.

When I examined him, six days later, the hemiplegia, although still evident, had to a great extent disappeared, and in a few weeks no trace of it could be detected. From that time to the present (April, 1897) his general health has been good, but the speech defect to be described has shown no marked change.

Word-deafness is almost total. The patient cannot be made to put out the tongue or perform the simplest movement without being shown what is expected of him, nor does he give the slightest sign of understanding simple questions concerning his name, age and residence. Salutations are the only expressions that seem to be understood; thus when I approach him and say, "How are you today?" he replies quite naturally, "Very well, I thank you." But on being asked, "Do you understand what I am saying?" he replies, "How?" and on the question being repeated as distinctly as possible, he says vaguely, "No, can't say that I do." Hearing for the watch and for ordinary noises is quite good.

Spontaneous speech is copious and fluent, and many words are correctly articulated, but many others are mutilated and many sounds intended for words are utterly in words are correctly articulated, but many others are mutilated and many sounds intended for words are utterly in talking quite well, his intonations are natural and the expressions of his face are intelligent. He cannot be made to repeat words said to him, but when he understands what is wanted he can count up to about eight. He cannot be

made to say the days of the week or the months of the year.

Acuity of vision is good. The patient easily finds his way about the city, recognizes persons at a distance and comprehends any visible change in his surroundings. Examination of the fields is difficult, but there seems to be no considerable defect; hemianopsia can certainly be excluded. On being shown a number of familiar objects, he very rarely ventures to name one, and if he does, the name is wrong. The aid of other senses makes no difference. When shown three familiar objects he cannot select the one named. His ability to use tools and other objects is not impaired, and he seems to have an intelligent understanding of pictures.

Comprehension of print and writing is lost, excepting his own name. Formerly he read a great deal, but now, on being urged to read, he replies with one of his few intelligible sentences, "I've lost all that." On being shown his name incorrectly written, "G. W. Long," he instantly recognizes it and points out the error, slowly writing a J in place of the G. On attempting to read it aloud, he reads, "J. W. Cullerdly;" the name "Grover Cleveland," he reads "Cupboard Crawford," and all his reading aloud is markedly paralexia. Nevertheless, he can correctly name the letters in many words, and after spelling a word he comes much nearer to pronouncing it, although he does not appear to understand it any better.

Attempts to read the time from a watch never succeed completely, although the minutes are sometimes read correctly. For example, the hands indicating 3.20, he says, "Twenty minutes after—" and then follows a jargon. Trying again, he says, "Fifteen minutes after—, fifteen minutes after three—. Don't know when in my life—," followed again by jargon.

He can write his own name correctly, but in carving it on a stick, he made it "J. W. Loog." Spontaneous and dictational writing are totally lost. Copying script is fair-

ly well done, with the occasional doubling or substitution of a letter. Words in Roman capitals are copied into script nearly as well. On being given a very simple problem in addition, he sets down some figures for the sum, but they are absurdly incorrect.

As there are many unsettled questions concerning aphasia in general, and this form in particular, and as the final appeal must always be to a comparison of clinical records and autopsy findings, I have studied and tabulated all the reports accessible to me of well-observed cases in which an autopsy showed the auditory centre for words, or its immediate neighborhood, to be damaged, without involvement of the motor-speech centre or of the visual centre for words.

It is now generally agreed that the auditory centre for words is situated in the posterior two-thirds of the first temporal convolution, on the left side in normal individuals, but on the right in left-handed ones. A more precise location is highly desirable, and this seems to have been furnished by Flechsig, who, at a period when the auditory fibres are conspicuous from having just received their medullary sheaths, has traced most of them to two transverse gyri on the inner or Sylvian surface of the posterior part of the first temporal convolution. In these two gyri, especially the anterior one, and in the sulcus between them, he, accordingly, locates the auditory centre. This is quite consistent with the autopsy records as far as they go, but most observers do not seem to have paid any particular attention to the inner surface of the convolution.

Altogether seventeen cases have been found which are regarded as fulfilling the necessary conditions. Five are included in Starr's list (*The Pathology of Sensory Aphasia*, Brain, July, 1889); the others, with one exception, have been published since. Cases in which the lesion extends backward no further than the supramarginal gyrus are included, because such an extension does not seem to alter the clinical picture of auditory aphasia; but cases in which

the lesion extended further backward have been rejected, on account of the uncertainty as to whether some of the symptoms have not been due to involvement of the visual centre, or of the fibres in immediate relation with it. In Case 9 the motor speech centre was found to be somewhat atrophied, but the case is included because Mills regarded this atrophy as a result of the long-continued disuse of the motor-speech apparatus which was secondary to the auditory lesions.

It must be confessed that some of these cases leave much to be desired as to fullness and accuracy in both the clinical and post-mortem records, and also that, owing to my distance from a large medical library, I have not had access to the original report in some cases, but have been compelled to rely on the best abstract I could find. Still, taking the cases together, with every due allowance for possible omissions or inaccurate statements, they furnish material for some valuable conclusions.

The cases are so tabulated that each one may draw his own conclusions from them, but a brief summary of the symptoms and of the writer's inferences from them may not be out of place.

Word-deafness existed to a marked degree in all these cases, although it should be noted that in Case 5 it was part of complete deafness due to the bilateral auditory lesion. There can be no doubt that word-deafness is the most characteristic symptom of a lesion of the auditory centre for words, and that it is a constant one.

Verbal amnesia, or impairment of the power to recall words to mind, is noted in eleven cases; in six nothing definite is said on this point, but in three of them (5, 8 and 15) it is safe to infer a degree of verbal amnesia corresponding to the degree of paraphasia, and in another (Case 3) the patient spoke so little that we would naturally infer considerable inability to recall words. In Case 10 spontaneous speech was said to be good, and if this was correct the recollection of words must also have been good, but the

CASES OF AUDITORY APHASIA WITH AUTOPSY.

REPORTER AND REFERENCE.	LESION.	COMPREHENSION OF SPEECH.	POWER TO RECALL WORDS.	SPONTANEOUS SPEECH.	IMITATIVE SPEECH.	READING ALOUD.	COMPREHENSION OF WRITING AND PRINT.	SPORTS-NEOUS WRITING.	COPYING.
1. Wernicke, <i>Der aphasische Symptomencomplex</i> , Breslau, 1874, Case 10.	Abscess of greater part of left temporal lobe. White matter of T ₁ softened; cortex normal.	Impaired.	Much impaired.	Paraphasia.		Paralexia.			
2. Kussmaul, <i>Ziemssen's Cyclop.</i> , Vol. XIV., p. 764.	Softening of T ₁ and T ₂ , right, mainly cortical. Patient left-handed.	Impaired.	Impaired.	Paraphasia.		Good.	Lost.	Lost.	
3. Giraudau, <i>Rev. de Médecine</i> , 1882, quoted in detail by Bernard, cited by Starr.	Sarcoma destroying posterior part of T ₁ and T ₂ , left, mainly cortical.	Lost.		Good, but spoke very little.	Lost.	Good (tests rather meagre.)	Good (after reflection.)	Good.	
4. Claus, <i>Irrenfreund</i> , 1883, f. 8, cited by Starr.	Softening of T ₁ and T ₂ , left; cavity occupying all of left temp. lobe except anterior two thirds.	Lost.		Fluent; no motor loss.					
5. Wernicke and Friedländer, <i>Fortsch. d. Medicin</i> , 1883, No. 6.	Gumma and softening of posterior two-thirds of T ₁ and T ₂ and all projection fibres of temp. lobe, left. Also of A. Sm and posterior part of T ₁ , right.	Deafness (owing to bilateral auditory lesion.)		Paraphasia.	Lost.				
6. Sepilli, <i>Rivista Sperimentale di Fren. e di Med. Leg.</i> , 1884, quoted in detail by Arndson, <i>N. Y. Med. Journal</i> , Feb. 14, 1885.	Softening of T ₁ , T ₂ and Sm, left; cortical.	Lost.	Much impaired.	Jargon paraphasia.	Fair.				
7. Hitzig, <i>Cong. f. Inner. Med.</i> , 1887, p. 166.	Softening of posterior two-thirds of T ₁ and T ₂ , left.	Lost.	Lost.	Paraphasia.					
8. Heubner, <i>Neurologisches Centralblatt</i> , 1889, p. 697.	Softening 27mm in diameter at junction of Sm and T ₁ , prolonged forwards 65mm between T ₁ and T ₂ , left, mainly cortical.	Lost.	Much impaired.	Lost.	Good.	Good.	Lost.	Paragraphia.	

9. Mills, Brain, Winter Part, 1891.	Softening T ₁ and T ₂ bilateral; left lesion older than right. First stroke 13 yrs, second 9 yrs before death: symptoms noted in interval.	Lost.		Paraphasia.			Good.	Good.
10. Pick, Archiv für Psychiatric, XXIII, 3.	Softening T ₁ and Sm, left, mainly cortical; T ₁ , T ₂ and insula, right.	Lost.		Good (slight paraphasia.)	Lost.		Good.	Good.
11. Ascher, Allg. Zeitschr. f. Psych., XLIX, p. 256. Resumé in Neur. Centr., 1894, p. 688.	Focal degeneration T ₁ , left. Diffuse degeneration of parietic dementia.	Much impaired.	Much impaired.	Paraphasia (used very few words.)	Good.		Lost.	Much impaired.
12. Fraser, Glasgow Medical Jour., 1893, p. 393.	Atrophy T ₁ and gyrus opercularis, left.	Lost.	Much impaired.	Paraphasia.	Lost.			
13. Leva, Virchow's Archiv. CXXXII, 2. Resumé in Neur. Centr., 1893, p. 786.	Cyst 3 cm in diameter occupying T ₁ and T ₂ , left. Slight focus of softening in foot of F ₂ , left.	Impaired.	Impaired.	Impaired.	Good.		Lost.	Impaired.
14. Mader, Wiener Med. Blätter, 1894, No. 8. Resumé in Neur. Centr., 1894, p. 700.	Softening T ₁ , T ₂ and posterior part of insula, left.	Lost.	Much impaired.	Jargon paraphasia.	Lost.		Lost.	
15. Worcester, Amer. Jour. Insanity, Oct., 1896, Case 3.	Disorganization of T ₁ , T ₂ and T ₃ in posterior two-thirds left, by bullet wound. Superficial track of ball over convolutions from temporal bone to apex of frontal lobe.	Marked word-deafness.	Much impaired.	Paraphasia.	Good.			Lost except for names of self and wife.
16. Worcester, l. c., Case 4.	Brain atrophied. T ₁ left very thin, yellowish and lathery. Convulsions elsewhere, normal in color, cortical layers distinct.	Lost.		Jargon paraphasia.				
17. Worcester, l. c., Case 5.	Softening of superior surface of posterior two thirds of T ₁ left, extending to the neighboring part of insula; also of foot of F ₁ , right.	Marked word-deafness.	Much impaired.	Paraphasia.				

EXPLANATION: T₁, T₂ and T₃—first, second and third temporal convolutions; F₁, F₂ and F₃—frontal convolutions; Sm—supra-marginal gyrus; A—angular gyrus.

report indirectly indicates a slight degree of paraphasia, which implies a corresponding degree of verbal amnesia. It is safe to say that verbal amnesia exists in about five-sixths of all cases of auditory aphasia, and it is very probable that careful tests would show it to occur in all cases except those in which the lesion is purely subcortical.

Spontaneous speech was lost in one case (8) and impaired (paraphasic) in thirteen; in Case 3 it was said to be good, but the patient spoke very little; in Case 4 it is described as fluent with no motor loss. Of Case 10 it is said that only occasionally a substitution of words occurred, indicating a slight degree of paraphasia.

The last case, being one of almost pure word-deafness and the only one with autopsy that I have been able to find, has a most important bearing on the theory of subcortical auditory aphasia. As is well known to all students of aphasia, Wernicke, Lichtheim and others explain the verbal amnesia, paraphasia, loss of imitative speech, paralexia, loss of comprehension of print and paragraphia, which are commonly associated with word-deafness, to be due to a loss of the normal influence of the auditory centre upon the other centres concerned in speech, and such a combination of symptoms is regarded as proof that the lesion is cortical. On the other hand, word-deafness not accompanied by the other symptoms is explained on the hypothesis of a lesion of the auditory fibres on their way to the auditory centre, the centre itself, although deprived of impulses from the ears, retaining its memories of sound and thus being able to exert its normal control over the other centres. The symptoms of Case 10 accordingly led Pick to diagnosticate a subcortical auditory lesion, but at the autopsy a cortical lesion was found, only slightly invading the white matter. Possibly a close examination of the auditory centre, as defined by Flechsig, with the white matter immediately underneath would have removed this disappointing discrepancy, but, until other cases of the

kind come to autopsy, we must accept this case as almost disproving the validity of the theoretical distinction between cortical and subcortical auditory aphasia.

The retention or loss of imitative speech is noted in ten cases. This function is worthy of careful attention, as its retention is the only thing that distinguishes transcortical from cortical auditory aphasia in Lichtheim's scheme. Case 8, if it stood alone, would be a brilliant illustration of the theory of transcortical auditory aphasia, for its symptoms were word-deafness, verbal amnesia, loss of spontaneous speech, loss of comprehension of print and paraphasia, with retention of imitative speech, just as the theory requires; and the lesion was so situated in the supramarginal gyrus and in the sulcus between the first and second temporal convolutions as not to destroy the auditory centre, but to cut off its communications upward, backward and downward. Hence if we suppose the association fibres from the auditory to the motor-speech centre to pass directly forward by way of the insula, they must have been spared, and everything about the case would be well explained. But in Cases 6, 11, 13 and 15, in which imitative speech is recorded as "fair" or "good," while the other symptoms were similar to those of Case 8, the lesion appears to have been cortical rather than transcortical. Moreover it is by no means certain that in Case 8 the path from the auditory to the motor-speech centre was intact, for it is only an assumption that this path passes directly forward—an assumption not justified by a study of the effects of lesions of the insula. On the contrary it seems to me more probable that this path takes a curved course, passing around the posterior extremity of the fissure of Sylvius, and that the "*Leitungsaphasie*" of Wernicke and Lichtheim is more likely to be found as a symptom of lesion of the supramarginal gyrus than of the insula. It must be remembered, too, that the retention of imitative speech accompanying word-deafness and paraphasia may be explained as due to a partial lesion of the auditory

centre itself. A word actually spoken stimulates the auditory centre more powerfully than the corresponding idea, hence imitative speech may be good when spontaneous speech is paraphasic; and the auditory centre can arouse the motor-speech centre more easily than it can the centres for ideas (proved by the existence of echolalia without understanding in various stages of incomplete development and impairment of brain function), hence in case of partial damage to the auditory centre imitative speech may be good when the understanding of speech is impaired.

Reading aloud was tested in ten cases. In four of these there was decided paralexia corresponding to the paraphasia; in Case 12 there was a slight paralexia (for difficult words only) along with marked paraphasia; in Case 10 reading aloud was good and paraphasia scarcely appreciable; in Case 3 the patient read short sentences correctly, and there was no paraphasia; in Cases 2, 8 and 11 reading aloud was good, but the patients had no comprehension of what they read, and there was marked paraphasia. This ability to read aloud, without understanding and without the ability to speak correctly, is in itself a very remarkable phenomenon, and is in strong contrast to the paralexia of other cases. In the present state of knowledge no difference between the lesions of the two groups of cases can be assigned to explain why the patients of one group can read while the others can not, and the facts seem to laugh at every theory of the process of reading. Considering only the cases without paralexia, we would naturally infer that in reading aloud the association impulses must pass directly from the visual centre for words to the motor-speech centre by a path that is quite independent of the auditory centre. Considering only the cases of paralexia, we would infer, on the contrary, that in reading aloud the motor-speech centre is dependent upon guiding impulses from the auditory centre, just as it is in spontaneous speech.

I think the true inference from all these cases is that

both in reading aloud and in naming a visible object two sets of association impulses pass forward from the visual centre, one set going directly to the motor-speech centre, exciting it to the utterance of words, but not always of the correct ones; another set going to the auditory centre, reviving the proper sound memories and exciting it to send a supplemental set of impulses to the motor-speech centre to insure the correct utterance. In some individuals the direct association between the visible words and the corresponding utterance has become so strong that it is of itself sufficient. In most individuals, however, the motor-speech centre will go astray, if not guided by the auditory centre, and in them an auditory lesion causes *paralexia*.

Possibly a similar explanation may apply to the occasional absence of *paraphasia*, and if so, it is evident, from the greater frequency of *paraphasia*, that the supplemental auditory impulses are more essential to spontaneous speech than to reading aloud. This is what we ought to expect. Letters being few and ideas many, each letter, by frequent repetition of the process, can become more strongly associated with its corresponding utterance than can each idea; and a centre, when excited by a sensation, sends out stronger association impulses than when excited by an idea.

The ability to understand print and writing was noted in eight cases. In five it was lost; in Case 3 it was said to be good after a little reflection, but does not appear to have been thoroughly tested; in Case 9, friends of the patient said it was good, but the physician did not have an opportunity to observe it for himself, and the testimony of an uncritical observer must be accepted with caution; in Case 10 it was undoubtedly good, corresponding to the ability to speak, read aloud and write correctly. Taken together these cases support the view of Wernicke and Lichtheim, that in the comprehension of written language the ideas are aroused by the auditory centre or, in other words, that the reader understands because he hears him-

self talk. I am convinced of the correctness of this view, but it must be admitted that there are anomalies which in the present state of knowledge do not admit of a satisfactory explanation.

Spontaneous writing was tested in eight cases. In five it was lost or impaired, the impairment being in the form of paraphasia. In Case 3, although the patient did not write much, spontaneous writing seems to have been better than spontaneous speech, quite contrary to the general rule in all forms of cortical aphasia. In Case 9 it was reported as good by the relatives, contrary to what we would expect, considering the existence of paraphasia and paralexia. In Case 10 it was good, in harmony with the correctness of spontaneous speech.

Copying was tested in only three cases. In Cases 13 and 15 it was impaired. Any decided impairment, not accounted for by the patient's general condition or by his distaste for copying what he does not understand, would justify the inference that there was more than an auditory lesion. In Case 13 a small lesion was found in the foot of the left second frontal convolution, and in Case 15 there was some involvement of the frontal lobe.

In summarizing the symptoms of auditory aphasia, we may say that word-deafness is constant; verbal amnesia and paraphasia are found in about five-sixths of the cases; probably a careful examination would show their presence in a larger proportion if not in all; imitative speech is sometimes retained, but more frequently lost or impaired, the difference probably depending upon the severity rather than upon the location of the lesion; reading aloud is often paralexia, but in many cases it is correct; comprehension of print was lost in the majority of cases tested, while in many of the severer cases no test seems to have been made; spontaneous writing was lost or paraphasic in most of the cases tested.

Does auditory aphasia constitute a fundamentally distinct type? Probably no one would for a moment

question the fundamental difference between it and the ordinary type of motor aphasia, but there are eminent authorities who do not recognize any radical distinction between auditory and visual aphasia. Thus Mirallié (*L'Aphasie Sensorielle*, Paris, 1896, p. 53), speaking also for Dejerine, contends that there is only one variety of sensory aphasia and that its symptoms include both word-deafness and word-blindness. He admits that either of these symptoms may predominate over the other, but still holds that both can be detected in every case, and that apparent differences of type depend upon differences of degree and not of kind. It seems to me that the general adoption of such a doctrine would be an unnecessary step backward. It is, of course, understood that a lesion limited to the auditory sphere may at first cause visual symptoms and *vice versa*, but indirect and temporary consequences should not be confused with the direct and permanent effects of a lesion. And it is also understood that a large lesion in the Sylvian region usually affects both the visual and auditory spheres, but it is the effect of small and limited lesions that should be considered. If any one doubts that there are two perfectly distinct types of sensory aphasia, let him compare such a case as the one just reported, characterized by word-deafness, jargon paraphasia, paralexia, loss of comprehension of print, agraphia, ability to copy Latin letters into script and absence of any visual defect, with such cases of visual aphasia as those reported by Wilbrand,² Lissauer,³ Dejerine,⁴ Dejerine and Vialet,⁵ Monakow⁶ and Redlich.⁷ In each of these cases in which the autopsy showed the visual sphere to be the only part of the language mechanism affected.

¹ Quoted by Freund, *Arch. f. Psych.* XX.

² *Arch. f. Psych.*, XXI., 1.

³ *Mém. de la Soc. de Biol.*, 1892, *résumé* in *Neur. Centr.* 1892, p. 373.

⁴ *Bull. Méd.*, Aug., 1893, *résumé* in *Neur. Centr.* 1893, p. 787.

⁵ *Arch. f. Psych.* XXIII. 3.

⁶ *Jahrb. f. Psych. u. Neur.*, XIII. 2. 3, *résumé* in *Archives de Neurologie*, Jan. 1896.

comprehension of speech was good, spontaneous speech was good, or only slightly impaired, and there was right homonymous hemianopsia. In each of these cases there was naturally an inability to comprehend print, and as this inability is generally found in auditory aphasia, it may seem to be true that word-blindness is common to both forms of sensory aphasia. It must be remembered, however, that, reading being a complex process, alexia may be caused in three quite different ways, by motor-speech lesions, by auditory lesions and by visual lesions, and it is only to alexia caused by visual lesions that the term word-blindness should be applied. The patient whose case I have reported in this paper gave abundant evidence that his visual functions, including visual memory, were in a condition of normal activity. It is not necessary to assume any degree of word-blindness to account for his alexia; he could not read because he was word-deaf for internal as well as external speech.

Disregarding minor arguments the reasons for insisting that there are two radically distinct types of sensory aphasia may be stated thus: Good observers have described cases of defect in the use of language due to a lesion of the angular gyrus or its immediate neighborhood, in which (after the temporary and indirect effects had passed away) no word-deafness could be detected, but permanent visual symptoms were clearly apparent; visual aphasia is the appropriate name for these defects. Contrasted with such cases are those forming the subject of this paper, whose lesion is in the posterior part of the first temporal convolution, and in which there is marked and persistent word-deafness without any persistent visual symptoms whatever; the speech defects of such cases are properly called auditory aphasia.

The impairment of all the ways of using articulate language, which is so evident in a typical case of auditory aphasia, is no doubt due to the fact that the auditory centre in the first language centre to be developed, and to its being essential to the normal development and cooper-

ation of the other language centres. The understanding of spoken words is the first linguistic acquisition in childhood, and this depends upon the formation of association tracts between the auditory centre and the various centres concerned in ideation. Utterance soon follows as an imitative reflex, and throughout life it depends for accuracy upon association impulses from the auditory to the motor-speech centre. Writing is an arbitrary representation of spoken words by visual signs, and reading is a reconversion of these signs into words as uttered and heard, and, as destruction of the auditory centre interferes with both understanding and utterance, it must also interfere with reading and writing.

DISCUSSION.

Dr. Philip Zenner, of Cincinnati—I will merely mention a case which was interesting to me, the post-mortem of which I saw last Friday. The patient was one who had Jacksonian attacks, and these were the only symptoms, with the exception of double optic neuritis; the attacks being chiefly in the right side of the tongue and right side of the face, though generally attended or introduced by sensory symptoms in the hand or face. Several times he had heard noises during the attacks, and one attack, described to me by his mother and himself, perhaps a day or two after it occurred, was distinctly an attack of word-deafness, in conjunction with the other symptoms already spoken of. For instance: I think he wanted a handkerchief, but he said, "Give me that shirt;" and when his mother said to him, "Here is a handkerchief," he evidently did not understand what she said. He admitted the next day that he had heard what had been said, but had not understood the words. At the autopsy a tumor was found lying in the lower part of the fissure of Rolando, evidently resting upon the first temporal convolution. This is the only case of word-deafness occurring as a part of a Jacksonian attack that I have seen, and I cannot recall having read the report of a similar case.

Dr. Charles K. Mills, of Philadelphia—I would like to say with regard to my case, to which Dr. Pershing has referred, and which was presented before this Association some years ago, that I still have the specimen in a pretty good state of preservation, and a short time ago glanced at it again. The retroinsular convolutions, which I understand to be the transverse convolutions referred to by Dr. Pershing, in this specimen are very much attenuated, as is also the whole of the

posterior third, or even more than a third, of the first temporal convolution.

The original lesion, the one which seemed to have caused the word-deafness, did not affect the retroinsular convolutions, but was situated in the posterior portion of the first temporal convolution and in the parallel fissure between it and the posterior portion of the second temporal.

In the general discussion of his subject I understand Dr. Pershing to take a position somewhat in opposition to that taken by Dejerine and others. With Dr. Pershing I believe that isolated lesions of the auditory centre, and similar isolated lesions of the visual centre, or a portion of them, will cause special and limited forms of aphasia, and will not result in an implication of all the forms of language.

Dr. Worcester, of Danvers, Mass.—I have had considerable experience with cases of this sort, and they are not infrequently admitted into hospitals for the insane, generally with an incorrect diagnosis, and I judge that not very infrequently the diagnosis is not corrected there. At last year's meeting of the American Psychological Society I read a paper embodying the results of my observations in full. I had at that time had four autopsies of cases of sensory aphasia. In two of them there was softening, and in one atrophy of the posterior part of the first left temporal convolution, and in the fourth there was a gunshot injury of the temporal lobe involving that portion of the convolution. In all these cases there was paraphasia, word-deafness, paralexia, and paragraphia—as far as there was ability to write at all; one patient had never learned to write. Two of them could sing tunes correctly, and the same was true of some other cases which have not yet come to autopsy. I have had altogether sixteen cases of this sort under my observation, and have had two autopsies since the date of the paper to which I referred, one of which was a case of softening.

In addition to this I had one quite interesting case of sub-cortical aphasia; it was the most complete case of aphasia that I ever saw. The man was a Swiss, and could not, so far as I could determine, speak a single word; the only articulate sound to which he gave utterance was something like "Sagasso," and he made use of this on all sorts of occasions. He had some comprehension of gesture, but none of speech. There was much dementia, and there was also complete uselessness of the right hand, but he was able to hobble about with the right leg. At the autopsy I found softening involving the whole of the external capsule, and the greater part of the nucleus lenticularis. The cortex seemed to be entirely unaffected.

Dr. Joseph Collins—Instead of the auditory speech centre

being spread out over too large a territory by the modern localizationalist and the student of physiology, it seems to me that it is allocated to an area so small that it is entirely incompatible with the grand importance which this centre has in conditioning speech development and maintaining the integrity of this function when it is developed. We have centred our attention too much on the supertemporal convolution of the left side, and have gone about looking for the smallest lesion of this area that would produce word-deafness. It would be more in line with the most legitimate facts and speculations in brain physiology to study the relation of the superior temporal lobe of the right hemisphere to the genesis of speech, and its preservation when developed. There are many facts indicating that the superior temporal convolution of the right side plays a most important part in these two things, and that the so-called auditory centre in the middle of the left supertemporal convolution is merely an executive centre, and is controlled by the same forces that determine righthandedness, be they tribal, sociologic or cultivated.

Dr. Pershing—The view Dr. Collins takes is the very one that I wish to combat in my paper; but the evidence will not be appreciated unless these cases, in which the post-mortem showed that the disease was limited to the auditory centre, are compared with the cases of lesion in the visual sphere, which I cited, though not at length. I think that if these groups of cases are put side by side my position will be substantiated.

In discussing the relative effect of lesions of the auditory sphere and of the visual sphere, it must be remembered that the auditory centre is the fundamental language centre; the first to be developed. All the later acquisition of the language depends on the activity of the auditory centre. As we learn to speak, or to read, or to write, we constantly call the auditory memory into use; therefore, if the auditory memory is destroyed, all these ways of using language are affected. Not so with the visual centre. If we may trust the reports of well-observed cases of visual aphasia, a lesion limited to the visual sphere does not cause word-deafness. This fact, I think, entirely disproves the views of Dejerine and Mirallié.

Dr. J. J. Putnam, of Boston—I had the opportunity to observe with care, some years ago, a case in which the patient was a literary man, and was able to make careful observations of his own symptoms. He eventually became hemiplegic, but at this time he was seized suddenly with word-blindness; he took down his Shakespeare, and found that he could not understand a single line. This passed away, but afterwards recurred. It was not, however, associated with any word-deafness at that time, certainly not to any considerable degree.

FOCAL CORD LESIONS.

LUMBAR POLIOMYELITIS ANTERIOR SUBACUTA UNILATERALIS.
TRAUMATIC CERVICO-DORSAL POLIOMYELITIS WITHOUT VERTEBRAL
FRACTURE.

By F. W. LANGDON, M.D.,

Of Cincinnati.

Case I. Lumbar Poliomyelitis Anterior Subacuta Unilateralis in a Man of Forty-four.

F. H. J., American, of German ancestry, aet. 44, is a salesman and married. He was admitted to the Cincinnati Hospital, neurological service, March 4th, 1896.

Complaint:—Loss of power in the left leg and foot, of five weeks' duration.

Family history is good; parents are living and healthy; no nervous disease is known. One daughter of the patient has "rheumatism." (This, on examination, appeared to be an angioneurosis due to lithemia.)

Personal History:—He has had most of the children's diseases in early life, except diphtheria. Had gonorrhea in youth. He denies syphilis, and presents no evidence of the same. Has been in good social and financial circumstances until recently, when he had much domestic and pecuniary trouble. He uses alcohol moderately.

The onset of the present complaint was gradual. Five weeks ago he first noted numbness and coldness in the left toes which was followed in a few days by weakness of the ankle and by foot-drop. He has had occasional cramps in the left sole and calf. There has been no tenderness to pressure, no nausea nor vomiting. The appetite has been good.

Present State:—Physique is good; height is 5 ft. 9 in.; weight is 160 lbs. He is of dark complexion, has gray eyes

and hair and looks well preserved. The heart, lungs and urine are normal. The radial arteries are elastic.

Left foot-drop is present. He walks with "high-action" step on the left side. The mental state and intelligence are good. There is no speech defect.

Cranial nerves:—Vision is good for ordinary reading. Visual fields are concentrically contracted. The pupils respond normally to light, accommodation and convergence. Smell, taste and hearing are good. There is no facial palsy nor anesthesia.

Trunk and Extremities:—Motor: muscular system generally is well developed and nourished. There are flaccid paralysis and moderate wasting of muscles of left leg and foot, both front and back. The power in the peroneal group is *nil*. He cannot flex ankle dorsally, but can flex the toes feebly. Grasp of right hand registers 80; of left 72.

Electrical Tests:—(See Chart I.) To faradism there is no response in the left peronei and extensors of the toes; sluggish response in the left tibialis anticus and calf muscles. To galvanism there is partial R. D. in all the muscles of the left leg; the contractions are very sluggish. $KC > AC$. Difference in poles is very slight. The muscles of the right leg and both arms responded normally to both currents.

Sensory: Tact and pain senses are normal everywhere. There is paresthesia to heat and cold over both legs, as per Chart II. He calls cold "hot" usually; hot "cold" rarely.

There is some defect of muscular sense in the left leg, foot and toes.

Reflexes:—Organic: There are no bladder nor rectal defects.

Myotatic: Knee jerks are active. $L. > R.$ Ankle clonus is absent.

Skin: Gluteal reflex on the right side is present; on the left is absent. Epigastric, abdominal, cremasteric, plantar reflexes are absent on both sides.

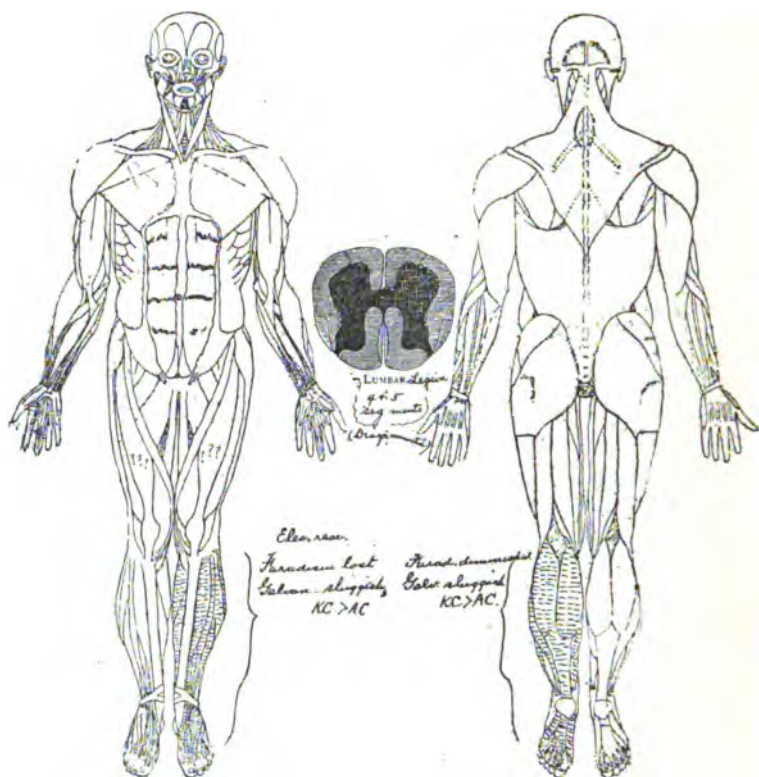


CHART I.

Lumbar Poliomyelitis Anterior Subacuta Unilateralis.
 == Paralysis, rapid wasting and R. D.

There is vaso-paresis over a small area (3×2 inches) in the left leg (see Chart II). This appeared as a dark purplish blotch, which later faded out, leaving a pigmented area.

Trophic: There are muscular wasting and R. D., as per Chart I.

Treatment consisted of massage, passive motion, galvanism, strychnia.

March 16th.—The patient can extend left toes slightly; paresthesia is unchanged. March 24th.—Area of thermo-

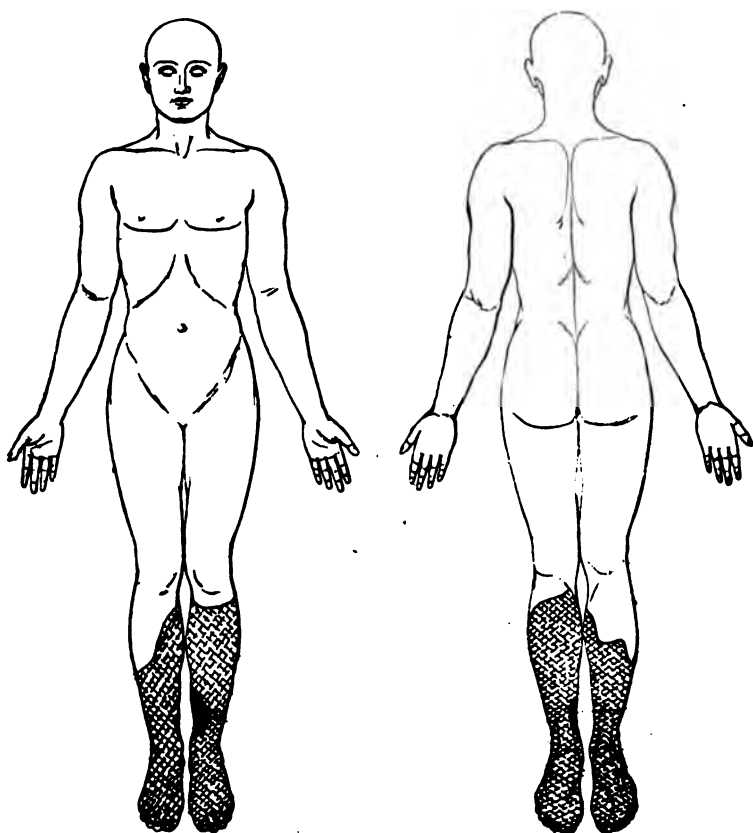


CHART II.

Lumbar Poliomyelitis Anterior Subacuta Unilateralis.

×× Area of Thermoparesthesia, March 4th, 1896. Calls cold "hot" usually; calls hot "cold" rarely. Tact and pain senses normal.

The dark-shaded oval area was purplish-red in color, and would probably have been the seat of a bedsore if subjected to pressure.

paresthesia is smaller; see Chart III. Knee jerks are normal and equal. April 13th.—Is discharged at his own request, much improved; with good use of both legs and feet. Sensation is normal. August 13th.—I saw him at my office to-day. He is practically well, and has no evidence of weakness in the left ankle to ordinary tests. No atrophy is evident. Electrical tests were not made. May

10th, 1897.—I met the patient on the street. He had no defect of gait and says he has perfect use of limb.

Comment:—This case is classified, in accordance with common custom, as a focal poliomyelitis anterior. There is every reason to believe, however, that the primary lesion was a thrombosis, to which the inflammatory process was secondary. Williamson,² and others, have adduced strong evidence in favor of this view, which is also in accord with modern ideas of the causation of infectious diseases and

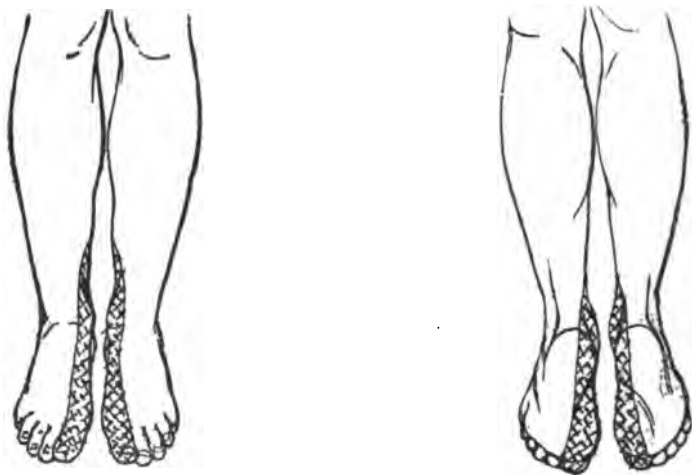


CHART III.

Lumbar Poliomyelitis Anterior Subacuta Unilateralis.
Thermoparesthesia, twenty days later.

softenings of the nervous system in general. Syphilis, as a cause for the thrombosis in the case here reported, would appear to be excluded by the absence of any history or objective evidence of that disease, as well as by the complete recovery without antisiphilitic treatment. The real cause, therefore, remains unknown.

²On the Relation of Diseases of the Spinal Cord to the Distribution and Lesions of the Spinal Bloodvessels. By R. T. Williamson, M.D., (Lond.) M.R.C.P., 1895. (Reprint with additions from the Manchester Medical Chronicle, 1894 and 1895.)

Case II. Traumatic Cervico-dorsal Poliomyelitis Without Vertebral Fracture.

H. P., colored, aet. 49, is a laborer. He was admitted to the Cincinnati Hospital, Sep. 25th, 1896, and was assigned to the surgical service of my colleague, Dr. N. P. Dandridge.

He was unconscious on arrival but became conscious twenty minutes later and gave a history of having fallen thirty feet into a cellar.

He complained of pain in the back and left shoulder. The shoulder was swollen, abraded and tender. A small scalp wound was seen on the vertex. There were no signs of injury to the vertebræ and no paralysis. Temperature upon arrival was 103.4 deg. Treatment consisted of an ice-cap to the shoulder.

Oct. 1st.—The swelling in the shoulder is about gone. Temperature is normal and he walks about the ward.

Oct. 3d.—Power is lost in the fingers of both hands.

Oct. 12th.—He was transferred to the neurological service.

Neurological examination, Oct. 12th, 1897, is as follows: He is a dark negro of average height, strong build and excellent muscular development. Pulse 64, regular, and there is no heart lesion. Urine is 1023, alkaline; contains no albumin, nor sugar, nor sediment.

His mental state is good, and he has no speech defect.

Cranial nerves: Vision is good to rough tests and there is no nystagmus. Pupils are moderately contracted; their dilatation is sluggish and extremely limited in extent. The pupils contract readily to light, accommodation and convergence. No other cranial nerve defects are noted.

Trunk and extremities—Motor: The muscles generally appear well nourished. There is no rigidity. Abduction and circumduction are present in both shoulders. Flexion at elbow is present and fairly strong in both arms. Extension is practically *nil*. Flexion and extension are very weak at the wrists; *extension* is *strongest*.

Grasp:—Dynamometer, R.O., L.O. Slight pressure of one finger at wrist easily controls triceps. The muscles of both forearms and hands are wasted and flabby. The costal portion of each pectoralis majora is also (see Chart

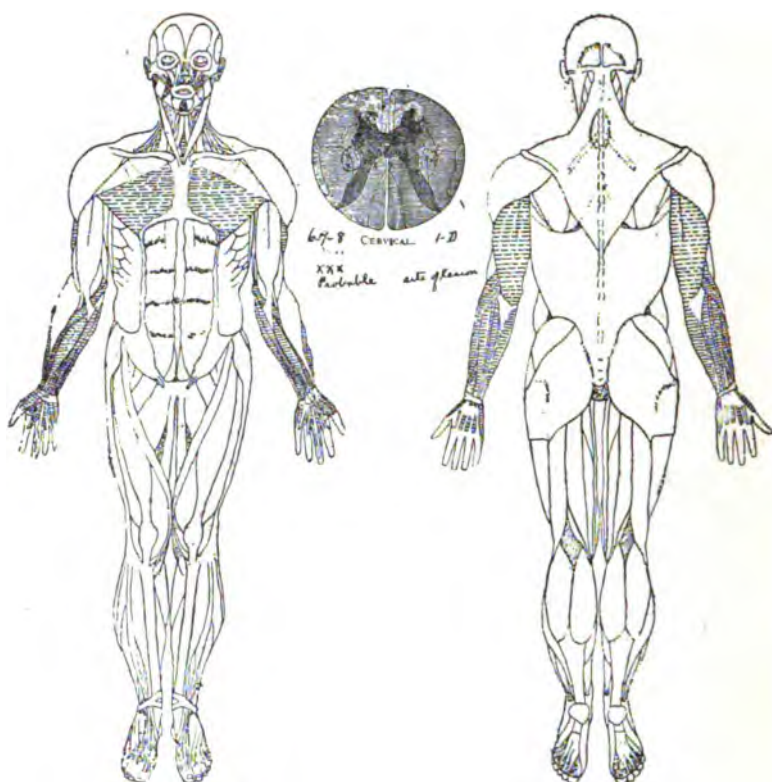


CHART IV.

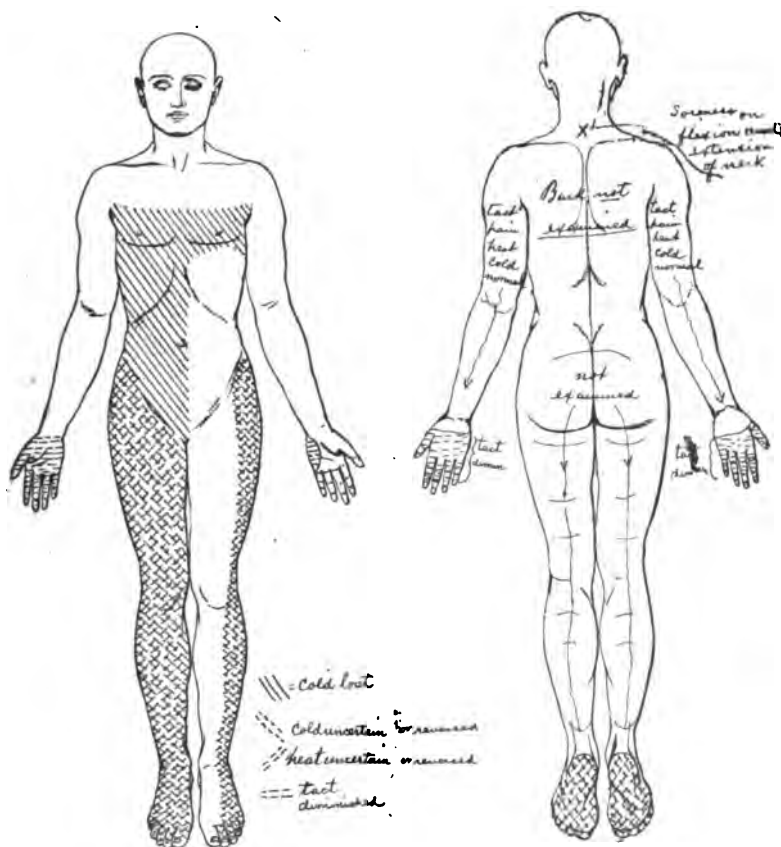
Traumatic Cervico-dorsal Poliomyelitis Without Vertebral Fracture.

= = Paralysis, rapid wasting and R. D. (Very sluggish response to faradism; no response to galvanism 15 M.A.)

IV). All movements of the lower extremities seem to be normal in extent and power.

Electrical Tests (see Chart IV.): Faradism yields a very sluggish response in the muscles shaded in the chart.

Galvanism: It is almost impossible to get any response in the same area with a bearable current (15-20 M.A.) (the skin is very dry and harsh). Response is good in the muscles not shaded.



F.W.I.

CHART V.

Traumatic Cervico-dorsal Poliomyelitis Without Vertebral Fracture.

- \\\\ Cold lost (heat preserved).
- \\ Cold uncertain or reversed.
- // Heat uncertain or reversed.
- == Tact diminished.

Sensory: Some soreness, not excessive, on flexion and extension of the neck. Tact and pain senses are diminished over a small area on the hand (see Chart V). There

are extensive defects of heat and cold senses as per Chart V. It was not thought advisable to turn the patient in order to test the back.

Reflexes—Organic: There are no defects of deglutition nor respiration, and no bladder nor rectal defects.

Myotatic: Jaw jerk is absent; elbow jerk is present and moderate on both sides. The wrist jerk is absent on both sides; knee jerks are present and active, R. > L. Ankle clonus on the right side is spurious; on the left side it is absent.

Skin: Palmar, epigastric, abdominal, cremasteric reflexes are absent on both sides. Plantar is present on both sides.

Vasomotor: Skin of forearms and hands is very dry and harsh.

Trophic: There is marked muscular wasting and R. D. as per Chart IV. No bed sores (now or later).

Diagnosis: Traumatic, focal (capillary?) lesion of 6, 7, 8, cervical and 1 dorsal segments, followed by poliomyelitis.

Treatment: Absolute rest; liquid diet; mercurial inunctions and iodide of potassium vigorously pushed.

Oct. 21st. Sensation: Heat and cold senses are uncertain (not lost) over entire area from nipple line to the feet anteriorly. Tact and pain senses are diminished on the hands, as before.

Nov. 20th. He is doing well, and has some power in the hands. The flexors and extensors of the hands respond feebly to faradism and galvanism.

Nov. 23d. He is improving rapidly; all sensations are good, and there is good faradic response in all the wasted muscles. The galvanic response is sluggish. The dilatation of the pupils in darkness is still sluggish and limited; contraction to light is active. The muscular power is improving.

Nov. 25th. He had a chill at 4 p.m., followed by a temperature of 104.6 degrees. Blood examination was negative. Quinine was given.

Nov. 26th. Temperature is normal.

Nov. 29th. Grasp: dynamometer, R. 17½, L. 15 (lower scale, numerically speaking.) Temperature varied from normal to 104 degrees, until Dec. 4th, when a fluctuating swelling was noted at left sternoclavicular joint.

Dec. 13th. He was transferred to the surgical service for operation. A purulent-looking fluid was evacuated from the left sternoclavicular region. Fever continued, and extensive mediastinal suppuration became apparent.

Jan. 1st, 1897. The patient died of pyemia. Autopsy was not obtained.

Comment: This case presents several features of interest aside from those of localization:

First.—The temperature of 103.4 degrees, a few hours after a severe injury was the only symptom referable to the spinal cord for eight days after the injury. The temperature fell to normal within a day or two, and did not rise again above normal for a month, when suppuration developed in the mediastinum. Five days after the fall the patient was up and about the ward, in his usual health and with no noticeable paralysis. Three days later (eight days after the fall) he noticed weakness in both hands, which rapidly progressed to complete paralysis, rapid wasting and R.D. of the muscles of hands, forearm, triceps and the costal pectoralis.

Secondly.—There appeared no evidence, subjective or objective, of any fracture of the vertebræ.

Thirdly.—The distribution of the lesion, as indicated by the symptoms, was limited almost entirely to the gray matter, as shown by the entire absence of bladder or rectal symptoms, as well as of leg paralysis or marked spastic symptoms. This would seem to the writer to indicate that the softer consistence of the cinerea, affording less support to its bloodvessels, had permitted the formation of multiple minute vascular lesions, which were followed by numerous capillary thrombi, insufficient in themselves to cause immediate symptoms, but capable of making their

presence evident after some days by exciting multiple foci of round-cell infiltration. This exudate was sufficient to cause pressure and abolition of conducting function, with only partial destruction of neuron bodies and axons, as is indicated by preservation of sluggish faradic response and by the rapid improvement in muscular power, the rate of improvement being too rapid to be ascribed to reproduction of axons. That complete recovery would have occurred had the patient lived, is, of course, not likely, since some permanent destruction must have resulted from consecutive sclerosis.

Fourthly.—The medico-legal aspects of the case are significant. In a case recently the subject of litigation, in which the writer was called to testify, there was good reason to believe that the cervical cord was the seat of organic changes, due to injury. The question of fracture, or non-fracture, of the vertebræ appeared to be one of great importance at the trial, the inference on one side being that if there were no fracture, the cord was not structurally damaged. This view is certainly not tenable, having in view the above and similar cases. Nor does it seem at all necessary at the present day from a neurological standpoint to prove a fracture of bone in order to establish organic injury to nerve centres.

The lapse of time (eight days) before marked symptoms of spinal injury, in the case here reported, is another point likely to give rise to forensic debate as to the connection between the injury and the myelitis.

Fifthly.—While there was no history nor marked evidence of syphilis as a factor in the case, yet mercury by inunction and iodide of potassium were pushed freely, with excellent effects, as would doubtless be the case in any event with such a recent exudate.

The actual cause of death, the mediastinal suppuration, is to be looked upon probably as the result of the fall, and was aided, perhaps, by the damage to the neurotrophic areas of the cord connected with that region.

Finally, the two cases here reported, taken together, would seem to throw additional light on the respective pathways in the gray matter of temperature and pain impulses.

It is well known that, in most cases of lesion limited to the gray matter of the cord, and especially in syringomyelia, temperature and pain conduction are affected together; tact escaping. In both the present cases, however, the temperature sense was notably impaired or lost, while the pain and tact senses were not appreciably affected. This would seem to indicate, not only separate pathways for temperature and pain, but also *considerable distance between them*; the temperature tract, for instance, would seem to cross to the opposite side in the anterior gray commissure, while the pain pathway would seem, as suggested by Turner³, to cross more posteriorly and to pass upward in the *substantia gelatinosa rolandi* of the posterior horns. This region apparently escaped damage in both cases here reported. Turner and Mackintosh, however, in a later paper,⁴ deny the necessity of assuming the existence of separate paths in the cord for the different sensations, considering that the dissociation of the temperature and pain senses is explicable by the hypothesis that "the more fundamental the form of sensation, the more extensive is the provision for its conduction in the cord." Hence, according to their view, temperature sense (vibration?), a late acquirement, is lost first, pain next, and touch last; the return in cases of recovery being in inverse order.

It would seem to the writer, however, that separate neuron-complexes for the conversion or elaboration of pain and temperature impulses may readily exist *in the*

³The Results of Experimental Destruction of the Tubercle of Rolando. By W. Aldren Turner, M.D., F.R.C.P. Brain, 1895, p. 231.

⁴Three Cases of New Growth with Cavity Formation in the Spinal Cord. By W. Aldren Turner, M.D., F.R.C.P., and Ashley W. Mackintosh, M.D. Brain, 1896, p. 331.

cord, without the necessity for separate *peripheral* pain and temperature-neurons. In other words, amongst the numerous short neuron tracts of the cord the spinal function may exist of converting (separating) heat and pain vibrations of the peripheral neuron into temperature and pain conduction respectively. It would seem to be rendered probable, by the two cases here reported, that this separation occurs in the cord; the lesions in the anterior horns and central gray matter accounting for the temperature loss; while the escape of the substantia gelatinosa rolandi and of the columns of Goll and Burdach explains the persistence of pain and tact senses.

My acknowledgments are due to Drs. G. C. Schaeffer and D. E. Robinson, the House Surgeons, for their careful notes on these cases; and to my colleague, Dr. H. H. Hoppe, for his courtesy in permitting use of later notes made during his term of service.

ANESTHESIA IN DISEASES OF THE SPINAL CORD.

By PHILIP COOMBS KNAPP, A.M., M.D.,

Clinical Instructor in Diseases of the Nervous System, Harvard Medical School; Physician for Diseases of the Nervous System, Boston City Hospital.

The clinical studies of Ross,² Thorburn,³ Starr,⁴ Head,⁵ Kocher,⁶ and many others, supplemented by the anatomical and physiological researches of Herringham,⁷ Paterson,⁸ and Sherrington⁹ have established the fact that the anesthetics or other sensory disturbances due to lesions of the spinal cord have a fairly definite distribution according to the particular segment of the cord involved. These areas follow the rule laid down by Herringham, that, of two spots on the skin, that which is nearer the preaxial border tends to be supplied by the higher nerve; and of two spots in the preaxial area, the lower tends to be supplied by the lower nerve; and of two spots in the postaxial area, the lower tends to be supplied by the higher nerve. That the exact boundaries of these areas are not yet established is apparent when we note the variations between

¹Brain, X., p. 333. 1888.

²Brain, XVI., p. 355. 1893. "A Contribution to the Surgery of the Spinal Cord."

³American Journal of the Medical Sciences, July, 1892. Brain, XVII., p. 481. 1894.

⁴Brain, XVI., p. 1, 1893; XVII., p. 339, 1894; XIX., p. 153, 1896.

⁵Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie, Bd. I., H. 4.

⁶Philosophical Transactions of the Royal Society, CLXXVII., 1887.

⁷Journal of Anatomy and Physiology, XXVIII., pp. 84, 169. 1894.

⁸Philosophical Transactions of the Royal Society, CLXXXIV., B., p. 641. 1893.

the diagrams of Thorburn, Starr, Kocher, and Head; yet, in spite of minor differences, there is a general agreement between them. Sherrington, moreover, has shown that the areas supplied by the different spinal roots overlap: that if one root be cut, there is no resulting anesthesia, because its area is supplied by fibres from the next roots above and below; so that in order to obtain anesthesia in the area of the first root, these other two roots must also be cut.

It is hardly necessary here to give a description of the different areas of anesthesia due to lesions of the different spinal segments, or to report more cases confirming the facts already established. It is sufficient to say that when we find anesthesia confined to these areas, we are warranted in diagnosing a lesion in the corresponding segment of the spinal cord, and the knowledge of this relation between definite areas of anesthesia and lesions of definite segments of the cord is probably the most important aid in the diagnosis of spinal disease that we have acquired in the past fifteen years. Anesthesias which have such a distribution, rendered familiar by the diagrams already referred to, may be termed spinal anesthesias. The distribution of anesthesias due to lesions of the peripheral nerves, peripheral anesthesias, was recognized long before this, and the diagrams of such distribution are to be found in most of the text books. In other diseases, notably in hysteria, the boundary of the anesthesia, instead of following either of the above types, may pass directly around the limb at right angles to its axis, and the anesthesia may occupy the area ordinarily covered by a sleeve, a glove, a stocking, or a sock, or it may cover the complete half of the body. Such a distribution, occurring as it often does in cases of cerebral disease, may be spoken of as the cerebral type, although the term, used only for convenience, should not be taken as denoting definitely the nature of the anesthesia. (Fig. I.)

Although we may thus classify sensory disturbances

according to their distribution as peripheral, spinal, and cerebral, and, although this classification is of distinct help in diagnosis, the distinction is by no means absolute. In one affection of the spinal cord, namely syringomyelia, it has been generally held that thermo-anesthesia and analgesia occupy "symmetrical segments on both the upper and lower limbs: on a diagram we might say that the patient has socks and gloves of thermic insens-

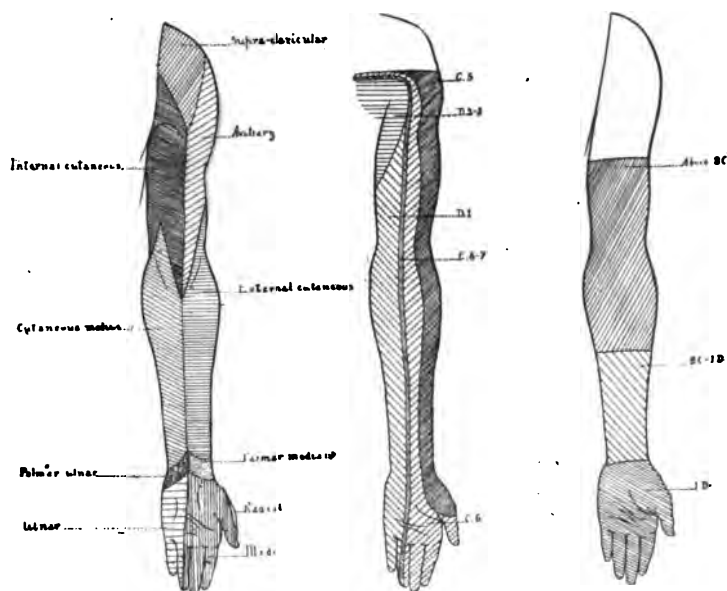


Fig. I.—Types of anesthesia: A, peripheral (from Hasse); B, spinal (root type of Chipault) (from Kocher); C, cerebral (medullary type of Chipault) (from Brissaud).

ibility."¹⁰ The accompanying diagram will show this clearly. The first case, M. C. (Figs. II. and III.), was reported by the late Dr. Jeffries,¹¹ and the first diagram is taken from his paper. The second observation was made several years later, and at that time there had developed a much greater degree of muscular atrophy in the arm. In

¹⁰Bruhl: "Contribution à l'étude de la syringomyélie," p. 20. 1890.

¹¹Journal of Nervous and Mental Disease, Sept., 1890.

consequence of a severe burn of the right arm, the elbow had been excised. The area of subjective numbness in the upper extremity, noted in 1889, had now become hypoesthetic, and there was an area which was hypoesthetic to pain, touch, and temperature on the anterior aspect of the lower abdomen and upper thigh, not coinciding with the area of subjective numbness of 1889. In 1889 the areas of sensory disturbance might have been regarded as of the spinal type, but in 1894 they seem more distinctly cerebral.

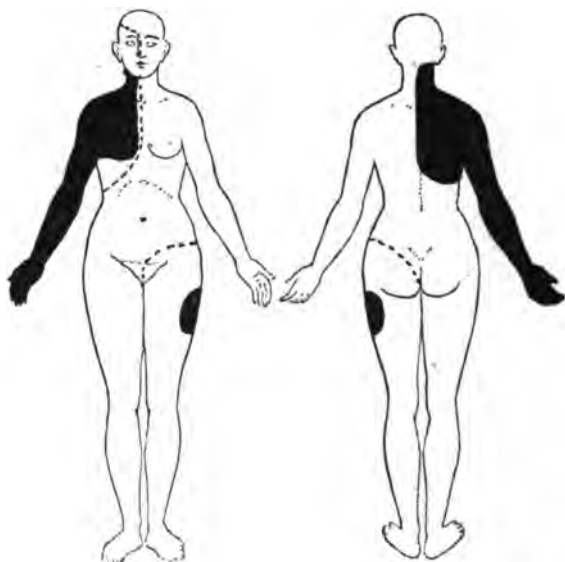


Fig. II.—M. C. Syringomyelia, 1889. Thermo-anesthesia and analgesia. Dotted line limits area of subjective numbness.

The second case, W. H. (Figs. IV.-VI.), was a man of thirty-seven, seen by me in 1892. He had a congenital spina bifida, operated on in 1891, and "spinal meningitis" in 1870. The trouble was of gradual onset. In 1883 he began to notice that the right leg dragged, and the weakness increased so that in 1884 he had to use a cane to walk with. The left leg also became affected, and in 1888 he had to use crutches. In 1884 he began to notice a loss of sensibility in the left leg and hand, and a year or so

later the right hand grew clumsy; he could not direct his movements, especially with the eyes closed, and the hand gradually became weak. He began to have rigidity in the limbs and pain in the back and thighs. He had some loss of control of the sphincters. The movements of the right arm were considerably impaired, but there was no ataxia. The movements of both legs were much impaired, the right more than the left. He could not raise the toes or the heel from the ground on the right side. There

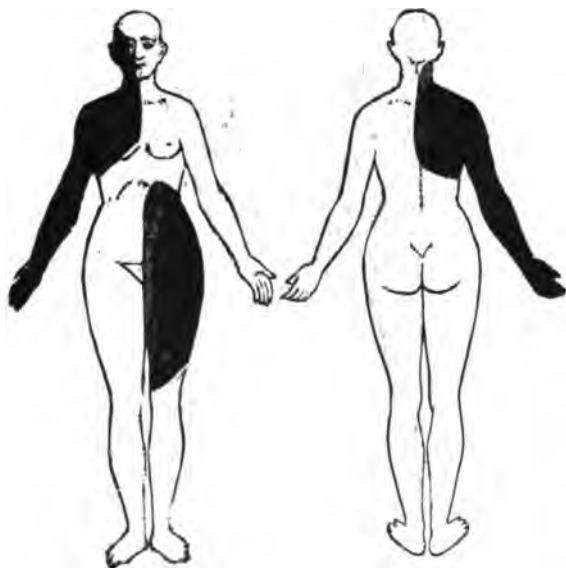


Fig. III.—M. C. Syringomyelia, 1894. Hypoesthesia to pain, touch and temperature.

was some extensor spasm in the legs, with a tendency to cross-legged rigidity. The knee jerks were not exaggerated. There was no noticeable atrophy and no change in the electrical reactions. The sense of position was impaired in the right forearm and right leg. The other sensory disturbances are shown in the diagrams. When first seen the anesthesia extended only to the level of the clavicle, but later it extended to the left side of the head.

From the earlier history and the first observation of

the loss of tactile sensibility this case might fairly be regarded as one which presented the phenomena of a unilateral lesion of the cord; the anesthesia being probably of the spinal type. The later history shows that the left side of the cord was subsequently invaded, and the distribution of the analgesia and thermo-anesthesia is clearly of the cerebral type.

As I have already said, it has generally been admitted

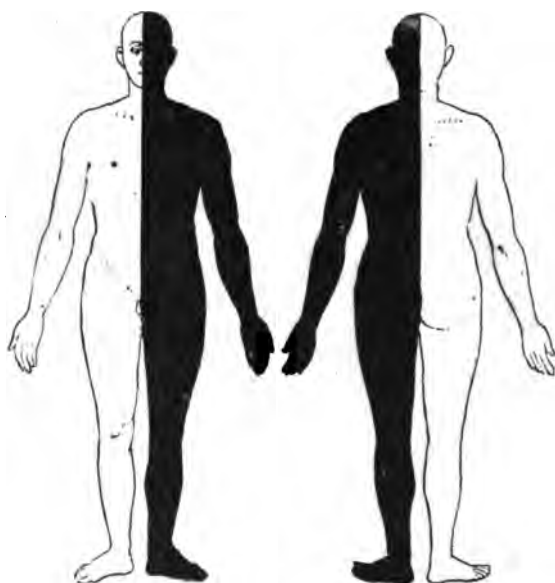


Fig. IV.—W. H. Siringomyelia. Anesthesia.

that the disturbances of sensation in syringomyelia followed the cerebral type, and many such cases might be collected, as, for example, to cite only a few of the reported cases where diagrams of these disturbances are given, the cases of Bruhl,¹² Roth,¹³ Lloyd,¹⁴ Souques,¹⁵ Schles-

¹²Bruhl: *op. cit.*

¹³*Archives de Neurologie*, Nov., 1887; March, July, Sept., Nov., 1888.

¹⁴*University Medical Magazine*, March, 1893.

¹⁵*Nouvelle Iconographie de la Salpêtrière*, 1891.

inger,¹⁶ Weintraud,¹⁷ Prince,¹⁸ Lamacq,¹⁹ Charcot,²⁰ Parmentier,²¹ Brissaud,²² Gilles de la Tourette and Zaguelmann,²³ and some of the cases of hæmatomyelia reported by Minor.²⁴ In many other cases where no diagrams are given, the descriptions indicate that the sensory disturbances were of the cerebral type.

In the two cases of my own which I have cited, I have indicated that the sensory disturbances were at times, or partially, of the spinal type. Max Laehr has recently

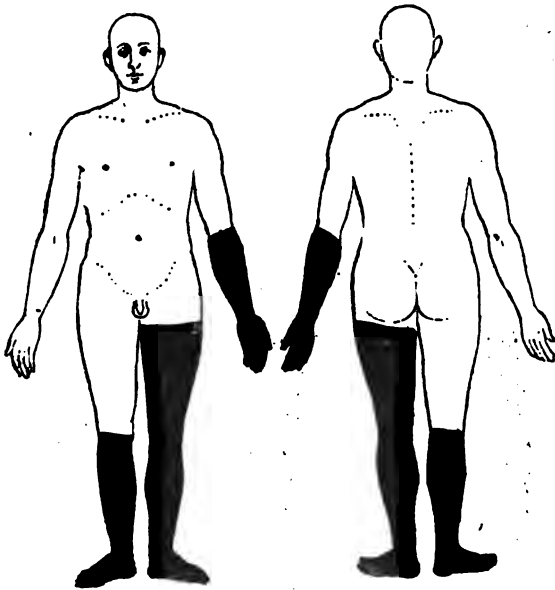


Fig. V.—W. H. Syringomyelia. Thermo-anesthesia.

made a very elaborate study²⁵ of the sensory disturbances

¹⁶Schlesinger: "Die Syringomyelie."

¹⁷Deutsche Zeitschrift für Nervenheilkunde, V., p. 383.

¹⁸"Dercum's Text-book on Nervous Diseases," p. 591.

¹⁹Revue de Médecine, April, 1895.

²⁰Charcot: "Leçons du Mardi, II., p. 506.

²¹Nouvelle Iconographie de la Salpêtrière, 1890.

²²Brissaud: "Leçons sur les maladies nerveuses," p. 191

²³Nouvelle Iconographie de la Salpêtrière, 1889.

²⁴Archiv für Psychiatrie, XXIV., p. 693.

²⁵Archiv für Psychiatrie, XXVIII., p. 773.

in syringomyelia, based upon seven cases of his own and a thorough search through the literature of the subject; and he claims that the distribution of the sensory disturbances in syringomyelia is not exceptional and of the cerebral type, as has been claimed, but that it follows the usual rule and is of the spinal type. A considerable number of other cases beside these which Laehr reports, among them the cases of Runge,²⁶ Eskridge,²⁷ and Patrick²⁸ in this country are clearly of the spinal type, while others

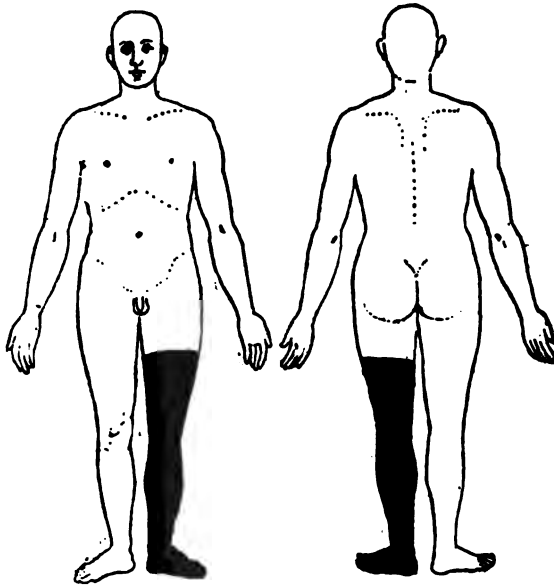


Fig. VI.—W. H. Syringomyelia. Analgesia.

show a distribution, like that of the first diagram of my first case or in the case reported by Dercum and Spiller,²⁹ which may possibly be of the spinal type.

It is needless to say that Laehr has not overlooked the many reported cases where the sensory disturbance was of

²⁶Journal of Nervous and Mental Disease, Jan., 1894.

²⁷International Clinics, Series III., Vol. 4.

²⁸Trans. Amer. Neurol. Assoc., 1897.

²⁹Amer. Journal of the Med. Sciences, Dec., 1896.

the cerebral type. In the first place it may be urged that hysteria may show sensory dissociations similar to those of syringomyelia, and that hysteria and syringomyelia may co-exist. While this may be true, it is certainly a begging of the question, however, to assume that hysteria co-exists simply because there is an anesthesia of the cerebral type with no other sign of hysteria, and in a part at least of the cases to which I have referred, there was no other symptom of hysteria except this cerebral anesthesia.

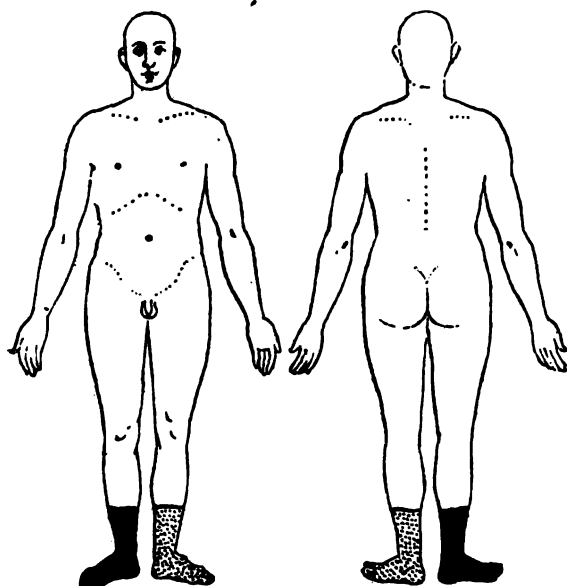


Fig. VII.—G. L. Tabes, Feb., 1897. Black, anesthesia; dots, analgesia.

Laehr's other explanation of the cases apparently of the cerebral type seems to me more valid. The sensibility to pain and temperature is less at the peripheral than at the proximal parts of the limb, as has been shown by Bernhardt³⁰ and Goldscheider.³¹ The disturbances of sensibility in syringomyelia are often only slight, and the spinal

³⁰Bernhardt: "Die Erkrankungen der peripherischen Nerven," I., p. 99, et seq.

³¹Archiv für Psychiatrie, XVIII., p. 659.

areas, which toward the central end are narrow stripes running longitudinally, often at the periphery expand. In consequence of this, the narrow strip above, whether hypoesthetic or normally sensitive in contrast with the rest of the arm, for example, may be overlooked, and the hypoesthetic or normal area in the hand may alone be taken into account. Although this explanation is much more probable than to suppose that the sensory disturbances are hysterical, the cases where these disturbances are

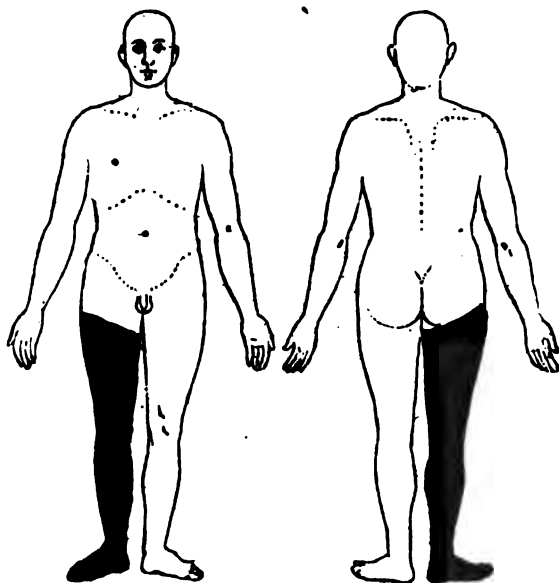


Fig. VIII.—G. L. Tabes, March, 1897. Anesthesia. Upper border not clearly defined.

clearly of the cerebral type have been reported by so many different trustworthy observers that we must acknowledge, at least for the present, that in syringomyelia the sensory disturbances may be both of the spinal and cerebral type.

Such a curious and apparently anomalous condition requires some explanation, and this has been attempted by Brissaud⁸² who, be it said, rejects the ideas that the sensory disturbances may be hysterical, and admits frankly

⁸²Brissaud, *op. cit.*, p. 219, et seq.

that they are due to the morbid process in the cord. Brissaud claims that the spinal segments themselves, at the cervical and lumbar enlargements, resemble the segments of the dorsal region. The lowest innervates the peripheral parts of the limb, the highest the proximal parts, and that the upper boundary of the anesthesia caused by a lesion of one of these segments is perpendicular to the axis of the limb—an anesthesia of the cerebral type. The spinal anesthetics are not due to lesions of the cord itself, but to

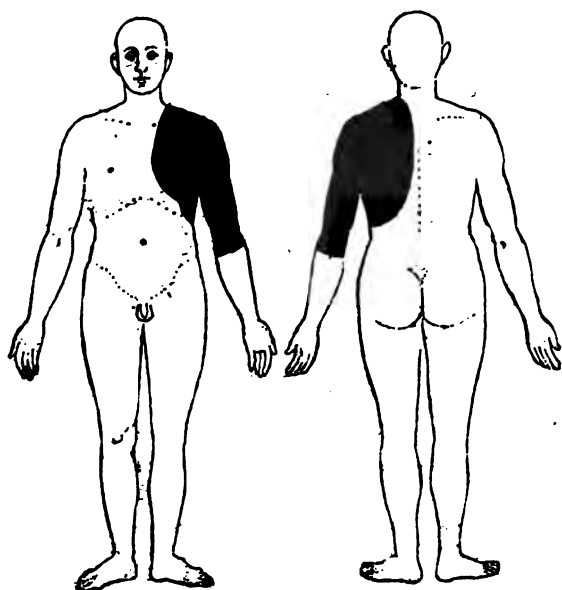


Fig. IX.—S. M. Tabes and muscular atrophy, April, 1896. Anesthesia and analgesia.

lesions of the spinal roots. This, he thinks, affords a valuable guide in spinal surgery: if the anesthesia be of the root, or, as I have thus far called it, of the spinal type, there is a chance of success; if the anesthesia be of the medullary (cerebral) type, it shows that the cord itself is involved, and that an operation would be of no avail.

Chipault,³⁸ accepting Brissaud's hypothesis, reported two cases of hemorrhage into the gray matter of the cord, where this medullary distribution of the anesthesia was

noted. In a subsequent study⁸⁴ of twenty-two cases of anesthesia in spinal caries, he found that seven presented anesthesia of the root type, six of the medullary type, two of the two types together, and seven of an indeterminate type. Chipault emphasizes the importance of this distinction pathologically, as indicating the seat of the lesion in the paraplegia of Pott's disease, in prognosis, since root lesions are less serious than those of the cord itself, and therapeut-

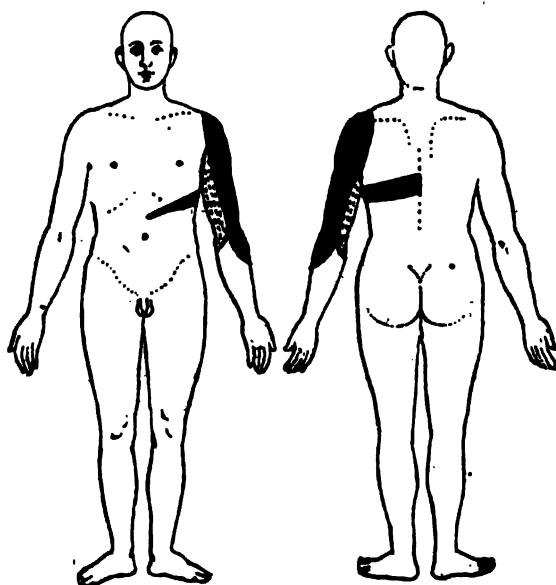


Fig. X.—S. M. Tabes and muscular atrophy, March, 1897. Black, analgesia; dots, hypoalgesia. No anesthesia.

ically, since if the cord itself be involved, the surgeon is not warranted in interfering.

The practical importance of this distinction between root and medullary anesthetics, if it can be maintained, is, of course, very great. The lesion in syringomyelia is most frequently central, which may explain why in so many cases the sensory disturbances are of the cerebral or medullary type. If the gliomatosis be situated more posteriorly in the neighborhood of the posterior roots, this may

explain why in some cases, as Laehr has shown, the sensory disturbances are of the root or spinal type.

Assuming the validity of this distinction, it would naturally be expected that in tabes the sensory disturbances would be of the root type. This has been shown to be the case by Laehr,⁸⁵ Chipault,⁸⁶ and Patrick,⁸⁷ and in a number of cases I have been able to confirm their observations, especially in regard to the narrow band of anesthesia on one or both sides on the trunk. Nevertheless, in a number of cases of tabes which have come under my observation

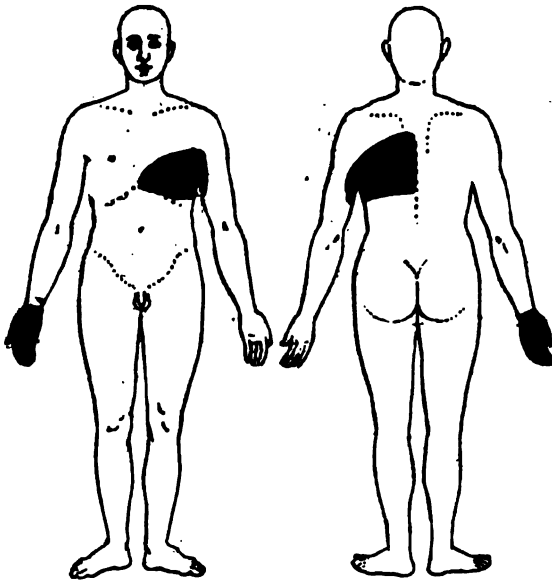


Fig. XI.—S. M. Tabes and muscular atrophy, April, 1897. Anesthesia.

this distribution of the anesthesia has not been noted, or, when noted, has not been constant.⁸⁸ In the anesthesia

⁸⁵Presse Médicale, Fed. 19th, 1896.

⁸⁶Revue Neurologique, May 30th, 1896.

⁸⁷Archiv für Psychiatrie, XXVII., p. 688.

⁸⁸La Médecine Moderne, N. 44, 1896.

⁸⁹New York Medical Journal, Feb. 6th, 1897.

⁹⁰Through the kindness of Dr. Pearce Bailey I was made aware of the similar conditions observed in a large number of cases of tabes by Dr. Bonar, the result of whose observations have been published in the Medical Record of May 22d, 1897, since this paper was read.

of tabes I have not infrequently found that not only were the boundaries of the anesthesia vague, as they are apt to be in spinal diseases, but that they varied from one period to another. The following cases will show the conditions of the sensory disturbances.

G. L. (Figs. VII. and VIII.), a man of fifty-two, had syphilis twenty-five years before. Ten to fifteen years before I saw him he began to have lancinating pains. About two years before I saw him he noticed that the legs were clumsy

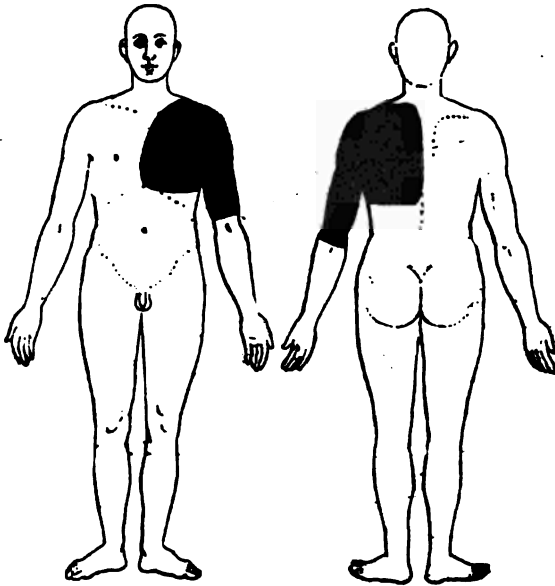


Fig. XII.—S. M. Tabes and muscular atrophy, April, 1897. Anesthesia.

when he tried to walk, and a little later the right hand and legs felt numb. Sexual power was lost. He swayed on standing with the eyes closed, the ulnar nerve was not sensitive on pressure, the knee jerks were lost, and the pupils reacted to accommodation but not to light.

S. M. (Figs. IX.-XII.), a man of thirty-seven, had acquired syphilis some years previously. In 1895 he noticed weakness and wasting in the intrinsic muscles of the right hand. At about the same time he began to have lancinat-

ing pains in the arm and chest. Some months later the left upper arm began to atrophy. In the spring of 1896 there was marked atrophy of the left deltoid, scapular muscles, biceps and triceps, and some wasting of the forearm, and considerable atrophy of the right thenar and hypothenar muscles. The pupils were unequal and im-

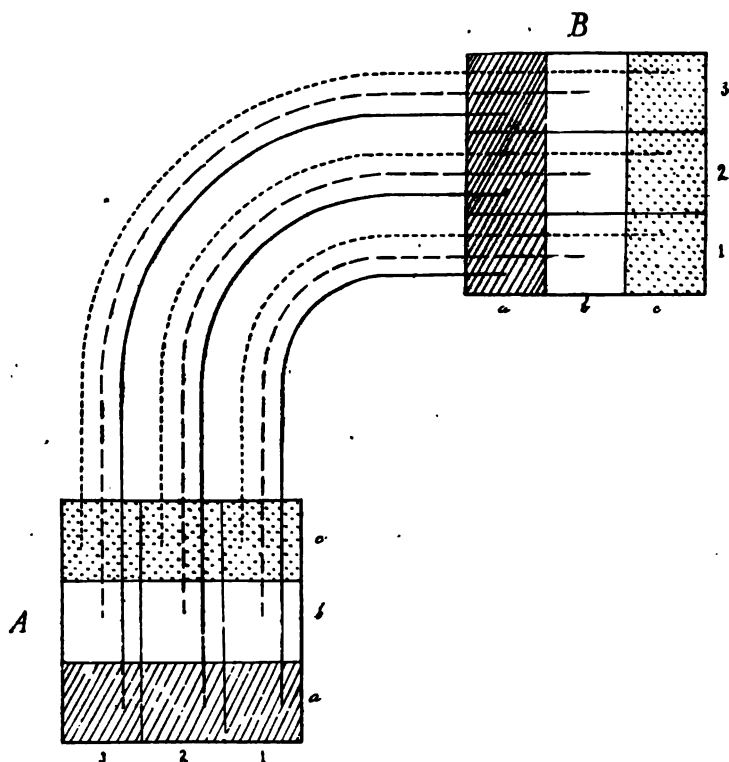


Fig. XIII.

mobile. The knee jerks were absent. The sensibility to pain, touch and temperature was diminished as is shown in the diagram. The atrophied muscles showed some fibrillary twitching and electrical changes in the most affected muscles. In March, 1897, similar conditions of atrophy existed. The optic nerves were beginning to atrophy. The

ulnar nerve was analgesic. There was no anesthesia, but some analgesia, clearly of spinal distribution. In April, the sensory symptoms showed a change, anesthesia re-appearing, partly spinal; partly cerebral in type, with analgesia which was distinctly cerebral. The conditions in other respects were practically the same.

Such cases, which, as I have hinted, are by no means exceptional, show that in tabes we do not always find that

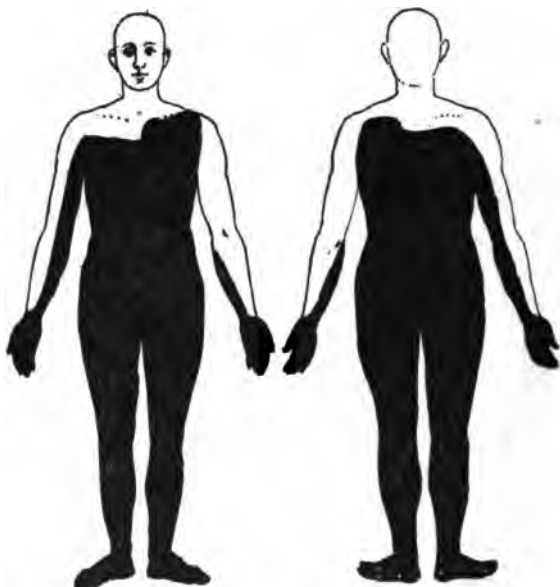


Fig. XIV.—E. R. Myelitis. Anesthesia.

the distribution of the sensory disturbances is of the spinal or root type, as would naturally be expected, if the hypothesis of Brissaud were accepted.

Ballet³⁹ has advanced another hypothesis which is, perhaps, more satisfactory. Ballet has constructed a diagram (Fig. XIII.), which attempts to explain why the sensory disturbances in spinal disease are sometimes of one type and sometimes of another. In this diagram A represents

³⁹Bulletin Médicale, Sept. 23d and 30th, 1896.

the upper limb, and B the gray matter of the cord. For simplicity we may suppose that the cord sends out only three roots, 1, 2, 3, each of which supply a longitudinal area of the limb, 1, 2, 3. A lesion which destroys the root, or the segment from which it issues, will cause an anesthesia in the longitudinal strip, 1, 2, or 3. In syringomyelia the lesion involves a considerable longitudinal extent of the gray matter. If we suppose that in each root there are fibres which rise at a greater or less distance from the centre of the gray matter and go nearer to or farther from the extremity of the limb, then a lesion running longitudinally in the cord in the regions a, b, or c, will be followed by anesthesia in the regions a, b, or c in the limb.

The following case may, perhaps, be explained by this hypothesis: E. R. (Fig. XIV.), a sailor, forty-seven years of age, with a probable history of syphilis, had a severe attack of dysentery in the East Indies in the summer of 1896. In October he noticed a loss of power in the legs, which gradually grew worse. Later he had pain in the legs, numbness and stiffness of the hands, a girdle sensation, and dribbling of urine. When I saw him in February, 1897, he was almost wholly paraplegic; the arms and hands were very weak; he had cystitis, difficulty in micturition, and general pains. He had had bed sores. The knee jerks were lost, and the muscles of the legs failed to react to faradism. There was diminution of tactile sensibility, as shown in the diagram, which seems to be of a mixed type. The hypothesis of a myelitis involving the cord transversely up to about the first dorsal region, and then extending up in a small portion of the gray matter for a segment or two more, would, on this theory advanced by Ballet, account for such a distribution of the anesthesia.

Although this scheme proposed by Ballet may satisfactorily explain such a case as this, or the varying conditions of sensibility noted in syringomyelia, it is hardly applicable to tabes, if we accept the present ideas as to the pathology of tabes. If we assume that the primary

lesion in tabes is a degeneration of the cell bodies of the peripheral neurons in the spinal ganglia, followed by a degeneration of the axis cylinder processes of these same neurons as they enter and pass upwards in the cord, then the sensory disturbance ought to be, as it so often is, of the root type. To explain other types of anesthesia we would have to assume changes in the gray matter of the cord beyond the simple degeneration of the sensory neurons. That such changes exist, at least in the more advanced cases, is well known, but their relation to the sensory disturbances is still problematical.

The relation of sensory disturbances to lesions of the spinal cord is, therefore, still undetermined. In many affections, notably syringomyelia, caries and tabes, we find such disturbances sometimes of the spinal and sometimes of the cerebral type. It is possible that sensory disturbances of the spinal type point more to lesions in the neighborhood of the posterior roots, and those of the cerebral type to lesions in the neighborhood of the gray matter, but that is still problematical. The scheme which Ballet presents affords a somewhat more satisfactory explanation than Chipault's original division into root and medullary types, but it is by no means conclusive.

(To be continued in the October number.)

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, June 1st, '97. B. Sachs, M.D., President.

A CASE OF INTRA-SPINAL TUMOR OF THE CERVICAL REGION, MISTAKEN FOR HYPERTROPHIC CERVICAL PACHIMENINGITIS.

Dr. Joseph Collins read a paper with the above title, and reported the following case: C. L——, a single man, thirty-six years of age, of intemperate habits, was admitted to the City Hospital on February 5th, 1896. About twenty years ago he had contracted a chancre, and this had been followed by the usual secondary manifestations of syphilis. He had been treated only during the continuance of the external manifestations. Nine months before admission he had begun to suffer from pain in the back of the neck, which at first was lancinating, but after a few months became paroxysmal. With this there had developed an inability to move the head easily. These attacks of spasticity coincided with the attacks of pain. About four months before coming under observation the head and neck had become fixed, and about two months later he had begun to lose the strength of the left arm. He complained of being extremely nervous, and of pains in the back and head. Examination showed that he maintained the cephalic extremity in a fixed position, and that, on turning to the right or left, the entire body rotated. The left hand was weaker than the right; the left knee-jerk was much exaggerated, and the triceps and wrist reflexes on that side were lively. The patient walked with a swaying movement. There was no muscular atrophy or deformity. The strength of the left upper extremity became steadily less, and the left hand became slightly flexed, but did not develop into a true "claw-hand." There was no impairment of the sphincters. The bowels had been very constipated, but on May 7th, diarrhoea set in, and was uncontrollable up to the time of his death, a month later. The patient complained almost continually

of severe pains in the neck and both upper extremities, and later of pains in the legs. As the disease progressed, the rigidity of the neck became less, and the extremities relaxed, although the deep reflexes were exaggerated. On June 8th he became delirious, and remained so until his death, one week later. At the autopsy the spinal pia was found to be distended and glistening, and, on puncture in the dorsal region, a quantity of clear cerebro-spinal fluid escaped, and the pia lost its distended appearance. From the first dorsal segment to the lumbar region, small white calcareous patches were found. The pia was neither thickened nor adherent, and the dura was translucent. The membranes of the brain appeared to be normal, as was also the quantity of intermeningeal fluid. A tumor of the pia was discovered on the posterior surface of the cervical cord, running from the lower border of the medulla oblongata down to the second cervical segment. It was 3.2 ctm. long, 2 ctm. in the widest part, and 1.5 ctm. at the narrowest end, which was directed upward. It was dark, reddish and firm. Microscopical examination showed it to be a spindle cell sarcoma with relatively enormous blood vessels. The brain was otherwise normal. There was an extensive pyelitis of the right kidney, and a less marked pyelitis on the other side. The liver showed interstitial changes, and the apices of the lungs, incipient tuberculosis. Nothing was found in the intestine to account for the diarrhoea.

The speaker said that the symptomatology of hypertrophic cervical pachymeningitis could be divided into two periods, viz.: (1) A period of pain and rigidity of the muscles of the upper extremity, indicating encroachment of the pathological process on the spinal roots; and (2) a period of paralysis, contracture, atrophy and deformity, particularly of the upper extremity, corresponding with the implication of the substance of the cord and secondary degenerative changes in the peripheral nerves. The pain was not unlike that complained of in the case just reported. It would appear that there were lacking certain symptoms which should be present to warrant a diagnosis of cervical pachymeningitis; on the other hand, the course of the disease, the unfolding of the symptoms, the character and intensity of the pain, should have suggested the possibility of intra-spinal tumor. Unfortunately no record was

made of the sensory disturbances. Dr. Collins said that even at the present time there was nothing like a consensus of opinion as to what constitutes the symptomatology of growths situated within the spinal canal; all that could be said was that the symptomatology depended chiefly upon the position occupied by the tumor, to a less extent upon the location, and to a comparatively slight degree upon the nature of the growth. Intra-spinal pressure counted for little or nothing, because of the entirely different relationship between the coverings of the cord constituting the lymph spaces and those existing between the brain and its coverings. Tumor symptoms were principally root symptoms. In the case just reported the root symptoms dominated to the end. The obstinate diarrhoea, he thought, was a symptom referable to the sympathetic system. Microscopical examination of the cord showed irregular degeneration of the columns of Burdach and slight degeneration of the columns of Goll, beginning at the lower level of the tumor.

Dr. F. Peterson asked if there were any other sympathetic symptoms beside the diarrhoea.

Dr. Collins said that he had not seen the patient until a few hours before death, and at that time such sympathetic symptoms as were present were looked upon as antemortem.

Dr. George W. Jacoby did not think there was a sufficient basis for a diagnosis of hypertrophic cervical pachymeningitis unless there were present the symptoms considered characteristic of that disease. The case just reported presented a pure symptomatology of a growth.

Dr. L. Stieglitz said that in the differential diagnosis of a tumor of the cord and pachymeningitis cervicalis, the asymmetrical character of the symptoms should be taken into account—the pain and paralysis existing in one arm only for some time. He could not agree with Dr. Collins that symptoms of tumor of the spinal cord were almost entirely root symptoms, for the exaggerated knee-jerk was an early symptom.

The president said, with regard to the symptomatology, that the root symptoms were generally the earliest ones, but other symptoms, such as the exaggeration of the reflexes, would appear relatively early if the tumor were sufficiently large to produce pressure upon the lateral columns. The curious thing about tumors of the spinal cord was that the unilateral character of the symptoms disappeared at a very early period, and did not necessarily denote any actual invasion of the spinal

cord substance; they were due simply to the effect of pressure. He had been interested in the sympathetic symptoms. It had been quite generally supposed at one time that the secretion of the kidneys was very much influenced by disease in the cervical portion of the cord, but experiments made years ago by several investigators, including himself, had cast some doubt on the correctness of that view. Eckhardt had found that interference with the ureters in the early experiments was apparently responsible for the inhibition of the renal secretion, and that the cord had nothing to do with it. The speaker said that pachymeningitis hypertrophica cervicalis had always been more or less of an enigma to him; he had seen very few cases corresponding to the description given by Charcot, and was in some doubt as to whether it represented a disease *sui generis*.

Dr. Mary Putnam Jacoby suggested that as a chronic pyelitis had been found in Dr. Collin's case, it was possible that a parenchymatous nephritis had been grafted upon it, and that the diarrhoea was simply a uræmic symptom.

Dr. Collins said that the point made by Dr. Stieglitz was extremely well taken. Spinal cord symptoms did not appear in pial tumors until towards the end, or when the tumor was of considerable size. Small tumors rarely made pressure on the cord itself because the pia was not bound down by diverticula, and hence had room to expand; but, as Dr. Stieglitz had very well said, in large and rapidly growing tumors, and of course, in tumors of the medulla itself, the reflex symptoms were very early and significant. He had been led to believe that the diarrhoea was a sympathetic symptom, from some recent investigations on the sympathetic nerve system. He believed that the sympathetic symptoms present in this case, of which diarrhoea was one, and the urinary complication was probably another, were due to a secondary degeneration or myelitic process set up after the disease had become well developed, and implication of the medullary substance of the cord from the pressure of the tumor.

KATATONIA.

Dr. Frederick Peterson read a paper founded on four cases of katatonia that he had personally observed at the Hudson State Hospital. Katatonia, he said, was a term applied to a certain group of psychical and motor symptoms which had often been considered as constituting a new and distinct form of insanity. He quoted the opinions of a number of authorities showing that katatonia had been looked upon as a clinical entity, as a variety of mel-

ancholia, as a form of circular insanity, as a species of hysterical insanity, and, by one author, as a tonic atonic mania. The symptoms were essentially variable, and exhibited a tendency to change from phase to phase, often returning to a previous one. The earliest symptoms were: abnormal sensations in the head, vertigo, insomnia, irritability, a certain amount of gradually increasing melancholia. The second stage might be ushered in by convulsions, or the patient might become maniacal. Muscular tension and spasmodic movements were present. Rhythmical movements were often seen, but were always under the control of the will. Sometimes there was a condition of religious excitement. Sometimes a tendency to talking and to acting theatrically, with, perhaps, a tendency to rhyming, would be observed. Catalepsy was often extreme and the muscles were generally in a state of tension. It was an extremely rare condition, he thought, presenting a symptom complex observed in many cases of insanity. The katatonic state could be distinguished from the insanity of pubescence by the fact that the delusions of the former were intellectual, depressed and varied indefinitely; in the latter, there were vague and stupid delusions. The chief diagnostic difficulty lay in differentiation from such cases of melancholia as manifested atonic or cataleptic states, but the rhythmic movements and tendency to repeat words and strike attitudes were important differential points. It was to be differentiated from hystero-epilepsy by the history rather than by isolated symptoms. From chorea it was distinguished by the fact that in chorea the movements were irregular and involuntary, and were not apt to exhibit such a variety of phases.

Dr. Peterson reported four cases—the only ones met with in a considerable experience, extending over fifteen years. The following case was typical of them all: B. R.—, female, thirty-one years of age, married, possessed of a common education. She was admitted to the Hudson River State Hospital in February, 1884. There was no hereditary history. The first evidence of mental disturbance was in August, 1883, two months after the birth of one of her children. She developed complete anorexia, lost interest in her surroundings, and, three weeks before admission, became suicidal. Menstruation was regular. The case was at first regarded as one of puerperal melan-

cholia. On February 11th she tried to beat herself against the bedstead, and claimed that she was being poisoned. She passed quickly into a condition of noisy excitement, and refused all food. She was markedly suicidal. She showed symptoms of exhaustion, and was fed by a tube for a considerable period. On March 15th she became markedly cataleptic; was absolutely silent, noticing no one, not even her husband. She began to wet and soil the bed. After a month or more she began to destroy her clothing and become filthy in her habits. In November, 1884, she began to cry out repeatedly, "Bring me to my children in New York," and this was repeated over and over again in a rhythmic manner, and more markedly on alternate days. After a while she began to rhyme with a series of unintelligible words. Meanwhile she was improving in her general condition. In the autumn of 1886 improvement was very evident, and after a short time she was allowed to go home. A recent report showed that she had recovered completely and permanently. Each one of the other three cases began as a case of melancholia, in which cataleptic conditions developed, and then this phase of verbigeration with rhythmical movements was added. One very prominent feature of all four cases was the melancholia. The verbigeration and rhythmic gesticulations were, perhaps, the most characteristic features.

His conclusions were: (1) that katatonia is not a distinct form of insanity; (2) that it has no true psychical character in its manifestations, and, hence, cannot be classed as a form of circular insanity; (3) it is simply a type of melancholia, and it is, therefore, not desirable to retain the name katatonia. Katatonic melancholia would be a convenient descriptive term. He was of the opinion that the prognosis in melancholia with katatonic symptoms was more grave than in any other form. The treatment was the same as for melancholia.

Dr. William Hirsch said that he had studied these cases while an assistant of Kahlbaum, who had originally described the disease. Katatonia was only one of a number of peculiar forms of insanity that Kahlbaum has created. The speaker said that he had very carefully studied these cases, as well as the records of the cases which Kahlbaum had himself diagnosed as katatonia, but he could never convince himself that katatonia was a distinct clinical entity. In none of these cases

could there be found a single symptom which was not, sometimes present in other forms of insanity. Kahlbaum claimed that katatonia gave a very favorable prognosis, yet a celebrated pupil of his had recently published the statement that the prognosis in katatonia was very bad. In 1874, Kahlbaum had asserted that katatonia was an organic disease, and had published certain pathological descriptions in support of this position. This seemed to be directly opposed to his estimate of the prognosis, and he believed that Kahlbaum had since abandoned these views.

Dr. William Leszynsky asked Dr. Peterson if those cases which showed cataleptic symptoms also exhibited symptoms of trance at the same time.

Dr. Peterson replied that they had not done so any more than the ordinary cataleptic.

Dr. Leszynsky recalled a case in his practice in which the patient had become cataleptic, and had remained in a trance state for three months. A certain surgeon had removed both ovaries from this patient with the idea of curing her. A curious feature was that she became conscious some time before death, and told him, in the presence of others, that she had been perfectly conscious of what had taken place in the past few months, and proved her statement by recalling certain incidents.

Dr. Hallock said that he had seen two cases of katatonia. One of these had passed into dementia; the other had been considered at one time to be a case of melancholia, but the later symptom had been of a hysterical order. The katatonic symptoms had finally passed off, and the patient had made a good recovery.

Dr. Rafel remarked that there must necessarily be great confusion, if, as Kahlbaum had stated, any one of the phases might fail.

Dr. Peterson, in closing the discussion, said that most of those who had followed Kahlbaum had looked upon the prognosis as very grave; some authorities had placed the recoveries at three per cent. Of the four cases reported in the paper, one recovered. The fatal cases passed into a condition of dementia, and generally died of tuberculosis, which was also consistent with the idea that it is a variety of melancholia, as this was a common termination in melancholia.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. S. SHIVELY, M.D., New York.
WM. C. KRAUSS, M.D., Buffalo, N.Y. A. STERNE, M.D., Indianapolis.
W. M. LESZYNSKY, M.D., New York.

ANATOMY AND PHYSIOLOGY.

DEATH BY ELECTRICITY.

Professor Julian Kratter, of Graz, experimenting on animals, with a view of studying the action of high tension currents, divided the results obtained into three classes. In the first class a secondary alternating current of 100 volts was used; in the second a primary alternating current of 1500 volts, and in the third a strong secondary current of 1926 volts. In general the danger of the electric current increases with its tension, but not in the same proportion, and the duration of contact determines the consequences, the action being very serious, if prolonged. On the other hand, death may follow, if the current is applied several times, even if the contact is not prolonged. Animals show a different degree of resistance to the electric current, guinea pigs and rabbits being very resistant. The symptoms observed are general tetanus, with cessation of respiration, while the heart actions continue. However, the heart may stop beating and death follow; but respiration always stops first. The primary action of intense currents is stimulation of all the centres in the medulla oblongata, and the sudden hyper-stimulation causes either temporary or permanent paralysis which may be fatal. In man the action of intense currents is evinced, in the first place, by loss of consciousness, in the second, by momentary nervous troubles or disorders of longer duration, such as the railway spine. At the points of contact, burns are found. In fatal cases, lesions are found, resembling those of suffocation, and small hemorrhages may be present in the meninges and the fourth ventricle. The author found in one case hemorrhages in the sheath of the pneumogastric nerve and symmetrical hemorrhagic spots along the cervical and dorsal vertebræ. These hemorrhages indicate, evidently, the course of the current, their distribution being due to the different resistance of the various tissues to the electric currents. The *resume* of Kratter's observations is that death by electricity is primarily due to paralysis of the respiratory centres, which is a particular form of asphyxia. (Wiener klin. Wochenschr., No. 18, 1896.) MACALESTER.

STUDIES IN SENSATION AND JUDGMENT. (Physiological Review, May, 1897).

TOUCH AND PAIN. Edgar A. Singer, Jr. (assistant Harvard Psychological Laboratory), experimented on the back of the hand and volar surface of the arm with a needle and a bristle for the purpose

of determining whether points which are sensitive to touch are also sensitive to pain. His experiments make the answer doubtful, but he thinks they point toward a discreteness of the end apparatus of touch and pain. He found that the thresholds of both touch and pain were altered by softening the skin with warm water, soap and glycerin, the threshold for touch being raised, though not at all points, while the threshold for pain was lowered, i. e., sensitiveness to touch was lessened while sensitiveness to pain was increased.

TEMPERATURE SENSE. His experiments confirmed Goldscheider's. Temperature spots were found to radiate from centres at which they are relatively numerous. The centres of the cold spots usually lie close to or coincide with the centres of the warm spots. The cold spots are on the whole more numerous; they reach more quickly and are more easily located. Both cold and warm spots seem to vary *inter se* in the strength of their reactions. Their sensitiveness differs at different times. By a slight pressure on the temperature spots (already located) warm and cold were experienced at the warm and cold spots respectively. Electrical stimulation had the same effect. In every temperature spot an analgesic point was found which would bear a heavy weight on a stimulating needle without producing pain.

INTENSITY OF SENSATIONS. This was found to vary considerably owing to the "subjective" factor.

JUDGMENT. It was shown that the greater the number of possibilities of judgment, the less the accuracy of the judgment. Every perception involved a combination with the stimulus of a concept prepared by past experience. Usually the number of concepts likely to be aroused by any stimulus is only limited by the context of events which the stimulus has entered; in the experiment we still lower this number, and in proportion as we lower it do we lower the threshold value of the stimulus.

He concludes, "Not only may a difference be noticeable or not, according to the way in which we define "noticeable," but, for any given criterion of noticeability, a difference may be noticeable or not, according to what we may mean by "difference." Mere difference may be noticeable at a point at which the specific kind of difference may be unnoticed. And, finally, with the same criterion of noticeability, a specific kind of difference may be noticeable or not, according to purely mental preparedness of the subject to receive it. With these facts recognized, the problem of psycho-physics awaits a re-statement."

CHRISTISON.

STUDIES IN THE PHYSIOLOGY AND PSYCHOLOGY OF THE TELEGRAPHIC LANGUAGE. By Prof. Wm. Lowe and Mr. Noble Harter, Indiana University, (Psychological Review, Jan., 1897).

Sixty operators were tested after Mr. Harter had spent eleven months in a preliminary investigation. The following are the principal facts developed: Highest sending record as far as known is 49 words per minute or $8\frac{1}{10}$ impacts per second. Subjective influences, as fear, anger, excitement, etc., have little effect on expert men other than to make them more fluent in the use of the telegraphic language. But in those not expert the emotions of fear, of anger and even of joy have the effect of paralyzing invention, so that only spasmodic or meaningless sound groups can be made, and every one recognizes that the man is rattled. Operators think in telegraphic terms. Those who work at night depend on their office call to waken them.

While an operator may detect an error or notice that a word was not appropriate in the connection used, and be able to suggest to the sender what the word should be, the language of the message as a

whole may have little or no meaning to him. There is a strong tendency to automatism with operators. Mr. S., a dispatcher, works daily with forty or fifty men, and states that after hearing four or five words he can readily recognize the sender or be sure that he is not one of his men. Many dispatchers claim that they can readily recognize a woman by her style of sending. The best time to learn telegraphy is eighteen years of age, and the maximum skill may be maintained until sixty-five years of age. Every operator shows personal peculiarities in his form of sending a message—by inflexions in experts, which increase in variations in proportion to expertness. At first the sending rate exceeds the receiving rate, but finally the receiving rate exceeds the sending rate as experience increases.

The operator hearing his own writing does not improve his power to receive in anything like the same degree that the hearing of other operator's writing does. Learners enjoy the practice of sending, but feel practice in receiving to be painful and fatiguing drudgery. As a rule, ordinary operators will not make the painful effort necessary to become experts. For many weeks there is an improvement which the student can feel sure of and which is proved by objective tests. Then follows a long period when the student can feel no improvement and when objective tests feel little or none. At the last end of this plateau, the messages on the main line are, according to the unanimous testimony of all who have experience in the matter, a senseless chatter to the student—practically as unintelligible as the same messages were months before. Suddenly, within a few days, the change comes, and the senseless chatter becomes intelligible speech. CHRISTISON.

THE MENTAL DEVELOPMENT OF A CHILD. By Cathleen Carter Moore (Psychological Review, Oct., 1896).

Mrs. Moore narrates and discusses the development of her child (a boy), during the first two years of its life, in a manner both precise and intelligent, and occupies 150 pages of a special issue of the Review with her subject. Nothing of the heredity or physical form of the child are mentioned. The following are the most noteworthy points given by her as she classified them. The observations of the first four were made by her husband.

Habits.—Within one hour from birth it sucked its thumb. It did the same at the end of its second year. After the first day it sucked its hands also, especially after long intervals, up to the eleventh week, after which it more or less sucked its thumb. Spontaneous movements often jerked its hand from its mouth up to the eleventh week when the jerks ceased. His aim for his mouth was very uncertain up to the eleventh week.

Hands and Arms.—During the first five days fingers were flexed in fists, and he slapped objects presented. Sixth day, fingers became frequently extended, but held near face. Twenty-ninth day, he would try to carry a presented finger to his mouth. Thirty-sixth day, he would grasp and hold clothing and carry a hand towards his face. Fifty-fifth day, he grasped at everything he came in contact with. Tenth week, he fingered things a great deal. Twelfth week, in his first attempt at reaching he fixed his gaze on the object, pursed his lips in attention and moved his hand graspingly towards it. Fifteenth week, hand uncertain and shaking in reaching. Seventeenth week, played with his own finger; did not try to get things beyond his reach, except by moving his body towards them. Twenty-first week, his feet became a favored plaything, and by the twenty-fifth week he objected to having them recovered. Twenty-sixth week, feet began to supplement the hands in feeling at objects. Twenty-seventh week, attempted to hold

with his feet. Thirtieth week, drummed on the table of his chair with a spoon or gum-ring. Thirty-second week, tried to grasp a fly with his whole hand. Thirty-third week, attended to two things at once—mouth his ring and grasp another object. He now quit playing with his feet, but used them for reaching and feeling. Thirty-fourth week, used his fingers separately. Thirty-sixth week, in reaching he used objects at hand. Thirty-eighth week, began to imitate with hands. Thirty-ninth week, used forefingers in feeling objects. Forty-fourth week, began to pick up objects with forefinger and thumb. Eighty-eighth week, forefinger used in pointing out objects. One hundred and fifth week, gave evidence of being right-handed.

Volition.—Fourth day, kissed when hungry, he turned his open mouth in direction of kiss. Seventeenth day, turned towards breast when hungry. Thirty-eighth day, gazed at visitor, and his body quivered by exertion. Twelfth week, tries to raise his body to a sitting posture. Seventeenth week, holds his head erect and tries to turn his body over. Twenty-sixth week, opens his mouth at the approach of a spoonful of water. Thirty-third week, recognizes things by resemblance. Thirty-fourth week, begins to discriminate and show preference. Thirty-eighth week, conscious imitation. Fortieth week, began to assist himself to stand. Forty-first week, drew objects to him instead of pushing them away. Forty-second week, repeats syllables and words. Forty-sixth week, creeping a habit; hunts for a person, lead by sound. Fifty-eighth week, acts previously forbidden by mother he would slyly do when she was not looking. Sixty-first week, walks alone. Seventy-third week, recognizes danger and recedes from it. Seventy-fifth week, shows obedience and previous discipline but displeasure at exactions by picking up two pins and bringing them to his mother, who, as always, takes them from him, and he is thereby displeased. Eightieth week, tries to give his doll a drink. Eighty-second week, conjoins after breaking. Ninetieth week, imitations more complex and frequent, and contrary actions increasing until twenty-fourth month as language becomes better understood. One hundred and first week, told purpose before acting. One hundred and fourth week, influence of association more manifest.

Inhibitory movements.—Third day, stopped crying by loud noise. Nineteenth week, would correct his motions. Thirty-ninth week, one good fall stops his farther kicking in the same way. Eighteenth month, would often cease to try when told to do so. Twenty-third month, when told to refrain, he would do so if not too long exposed to temptation.

Automatic Movements.—They were frequent during sleep, night and day, until the sixth week, when they gradually almost entirely ceased. Twenty-third week, a noise which heard once before awakened him, now only evoked a cry in his sleep without opening his eyes. Twenty-fifth week, could readily find the breast and suckle while asleep.

Emotion.—First day, cried at discomfort. Sixth day, smiled at comfort. Seventh day, smiled at father four consecutive times and simultaneously moved its arms. Tenth day, tears observed. Seventeenth day, fretting cry in discomfort. Forty-sixth day, laughed. Sixty-first day, pursing lips with attention. Tenth week, rapid motion of limbs in excitement. Twelfth week, urine observed to be longer retained under exciting or normal experiences (six hours at longest). Ninth month, excitement interfered with evacuation of fæces also. Nineteenth week, writhing and twisting body expressed delight. Twenty-fourth week, kicking, laughter and screaming expressed pleasure. Twenty-fifth week, fretting and scolding at disappointments and

vigorous movements of legs and body in proportion to excitement or strength of feeling. Thirtieth week, frowned when annoyed, movements rhythmical. Thirty-third week, smile changes and becomes more expressive. Forty-third week, delight expressed by piercing scream and active movement of limb. Forty-sixth week, delight expressed by shiver, noticed only for six weeks more. Fifty-fifth week, mischievous look. Seventy-eighth week, shyness by hiding head; squarely open mouth in crying becomes more frequent. Eighty-seventh week, struggled and hit when opposed; this lasted only a few weeks. End of second year, pleasure expressed by stamping, trembling hands, sparkling eyes, and running around and around.

Notes.—Muscles of face more quiet in a profound sleep than in a light sleep. First smiles were produced by muscles around the mouth and not perceptibly around the eyes. They were extremely evanescent and occurred on peacefully dropping to sleep; occasionally at persons. Finally the whole face, including the scalp, united in producing a smile. Smiles never occurred when the child was known to be in pain. At birth there were well-defined movements expressive of pain, but none for pleasure until the dawning of intelligence. For displeasure there were few modifications, but for pleasure there were many in the course of development.

Reflex Movements.—These were not observed systematically. They were seen the first day. All gradually ceased to respond to their stimuli, except such as were instinctive, or modifications, or additions to instinctive movements—direct accommodations to environment; and those that were instinctive plus spontaneous movements.

Sight.—First day, eyes opened by only a narrow crack; balls constantly rolling open or shut; pupils hardly affected by strong light. Second day, looked intently at a bright object and followed its movement. Third day, eyes wide open but not in focus; turned from one object to another. Eighth day, eyes first seen in focus; looked at surroundings for half an hour. Tenth day, eyes often in focus, followed objects moving to and fro. Forty-seventh day, looked with interest at things. Sixtieth day, looked at strange faces seriously, but smiled at those familiar. Twelfth week, would remain quiet an hour watching trees sway in the wind. Fiftieth week, made grimaces at his own reflection in a mirror and ceased when he saw by reflection that he was observed. Fifty-eighth week, recognized a person he had seen but once for a few moments three days before, but by whom he had been hurt. One hundredth week, he never showed any preference for colored pictures over uncolored pictures.

Hearing.—Second day, first definite reaction to sound—it several times stopped crying when its father began to whistle. During the first common sounds were comparatively ineffective. Fourth day, frequently looked at father when spoken to by him. Seventh day, looked intently when spoken to. Seventeenth day, peculiarly sensitive to sounds—was restless and apparently not well. Twentieth day, lay still for fifteen minutes when some one was singing to him. Thirtieth day, turned his head in the direction whence sounds proceeded. Seventeenth week, evinced pleasure in an action song. Twenty-first week, the slightest sounds seemed to interrupt his meals; he looked in their direction. Twenty-sixth week, voices which formerly startled him and made him cry, now only create curiosity. Twenty-seventh week, location of sounds fairly well established, except those from behind his head. Thirty-eighth week, he drummed continuously with some hard object on the table of his chair. Forty-first week, began to distinguish among spoken words. Fifty-fifth week, he understood a little language

and possessed some words of his own. Ninety-eighth week, he could refer all the noises of the house to their sources.

Touch.—Sixth day, begins to use palm of hand by pressing it against his mother's breast while sucking. To be soiled or wet causes him to cry. Eleventh week, begins to feel objects. Sixteenth week, plays with his own fingers. Twentieth week, sometimes uses his feet to feel with. Twenty-fourth week, objects to covering on his feet. Twenty-seventh week, feels objects with hands and feet, and then puts them to his lips. Flies waken him if they walk on his face. Thirty-ninth week, forefinger tip used to feel small objects. Fifty-second week, localizes pain well—puts hand on the hurt.

Mental Development.—Involuntary attention established immediately after birth. Some representations were established by the ninth month, but not until the second year were fairly distinct images possessed. During the second year associations were gradually formed connecting mental image with mental image, but prior to this there was no faculty of voluntary recall. Associations of similarity began to occur about the fortieth week. Associations of simple contiguity predominated at all times. In the ninety-eighth week every act of the child, during the greater part of a day, was found to be connected with such association. The suggestion of object by object was not a common occurrence before the seventy-first week. His perception of likeness was not the reverse side of the perception of difference. Reconstructions independent of original contiguity began to occur about the close of the second year. He had some class ideas but no abstract ideas pertaining to his concrete words.

Late in the second year language became a channel for the conveyance of thought, but when a representation had been evolved, it was concrete and limited. Generalization and induction as well as inference occurred. An embryonic induction was thought to occur when the child refused to reach for an object whose distance from him was greater than the length of his arm. The higher forms of thought were developed with the more complicated movements and existed in embryo before abstraction was to any extent possible, and inference, induction, generalization and reconstruction were mental habits in the same sense that sitting, creeping, etc., were habits, and that each mental habit was developed by a method similar to that found in the development of a bodily habit.

Language.—First sound, the expiratory *a* in crying. After the eighteenth week the variation in tones slowly disappeared, and the voice gradually settled into a clear falsetto. Twelfth week, he began to use his tongue and had made about all the sounds of the language. Thirty-sixth week, he acquired the habit of repeating a sound of his own upon hearing it uttered by another person. Thirty-eighth week, he began to associate a few words with persons and objects. Forty-second week, conscious but unintelligent imitation became habitual. Sixty-fourth week, the first verb (*gone*) made its appearance. Sixty-sixth week, the first sentence was uttered (*papa gone*). Ninetieth week, the first action: word proper came into use (*sit down*). At the end of the second year the child had acquired 475 words as follows, viz.: Nouns, 306; verbs, 92; adjectives, 38; adverbs, 14; pronouns, 11, and of prepositions, interjections and conjunctions, 14 in all.

In the early days of language development, each word whose faulty pronunciation had been laboriously acquired replaced for a time the old words which for several days were scarcely heard. The application of a word was extended slowly and by degrees. At the close of the second year the child invariably asked the name of a new object.

CHRISTISON.

NEUROPATHOLOGY.

THE VARIABLE CHOREA OF DEGENERATES. (*Revue Neurol., Presse médicale.* Abstract in *Jour. de Méd. et de Chirurg. Prat.* Oct., 25th, 1896.)

Under this name Brissaud describes a chorea which has no uniformity in its manifestations or regularity in its evolution, and no constancy in its duration. It comes and goes, alternately increases and diminishes, ceases all at once, reappears without premonition, and again disappears; the movements are now sudden, again they are slow, and they are without any particular localization.

It always develops in degenerates in the strict sense of the term. Its appearance is due to the influences of various causes, and it begins so gradually that it is difficult to indicate the commencement. Indeed, until the trouble has attained a degree undeniably pathological, it is ordinarily considered to be a simple muscular "caprice" of no particular significance.

Once established, the syndrome is characterized by involuntary movements which would commonly pass under the term "nervous movements." They are more or less rapid, and affect particularly the arms, shoulders and face, are always involuntary and usually pronounced in degree and frequency in proportion to the general nervousness of the patient. But it is important to note that these grimaces and gesticulations are inconstant from one day to another, and even from one moment to another; they may disappear for several days, and suddenly reappear when the patient has been apparently cured. They may be to a certain extent voluntarily checked, but the efficiency of will power is of only short duration. In short, the affection is variable in every respect.

Brissaud affirms that the cases are frequent, but he reports only four in detail. In the fourth case the trouble developed in a typical degenerate at the time of adolescence, and lasted four years. At the end of this time it disappeared completely. This sudden disappearance is of capital importance in regard to the prognosis, and also in regard to the diagnosis, as it is one of the principal signs by which it is distinguished from Huntington's chorea.

Variable chorea can be confused with but two affections: ordinary minor chorea and Huntington's chorea. *Paramyoclonus multiplex* and rhythmic chorea, which is always a symptom of hysteria, have nothing in common with the affection under consideration. The diagnosis from ordinary chorea is not difficult, as the latter disease is a well-defined entity, in evolution and symptomatology. Furthermore, variable chorea presents two characteristics which are wanting in chorea minor. First, the multiplicity of form shown by the movements; second, the fact that in the former affection the movements may for a moment be controlled by the will.

The chronic chorea of Huntington is an incurable malady, and lasts a life-time without intermission. It is chronically progressive, not only as regards the muscular manifestations, but also as regards the accompanying psychic degeneration, which latter is wanting as an essential symptom of variable chorea. In contrast to this unvarying chronicity and progression, variable chorea is never the same for two days, or even two hours, together. He presents intermissions without apparent cause, and, as before noted, is uncertain and variable in every way. Huntington's chorea is apt to affect in particular this or that group of muscles. This may also apply to variable chorea, but it is never constant to the same muscular group, quickly and without apparent cause changing from one to another.

Brissaud relies upon this general variability to constitute the entity of a well-defined muscular neurosis. He considers it to be one

of the transient affections frequently found in the subjects of degeneracy.
PATRICK (Chicago).

DÉBILITÉ MENTALE ET TREMBLEMENT.

Labbé (*La Presse Médicale*, No. 33, 1897) describes a case of tremor, associated with epileptiform attacks, athetoid movements, and stigmata of degeneration, a woman of twenty. At an early age she had a right hemiparesis, and at four months old commenced to have convulsions of an epileptiform character, which recurred several times a month, until she was seventeen. She was then free for three years, but lately has begun having convulsions again, and now they are more hysteroid in form, though the stigmata of hysteria is not present. There is now but little difference in power between the two sides of the body, and their reflexes are about equal. The tremor, which has existed since childhood, affects the whole body, but is much more marked on the right side, especially in the right arm and hand. It is increased by emotion, and to a less degree on voluntary movement. The tongue trembles but little, the eyelids not at all. There are also athetoid movements in both upper extremities, more marked on the right. Mental power is low, but the patient is not an imbecile. From a study of this case the author thinks that the tremor cannot be referred to the presence of the usual causes, but is inclined to consider it hereditary. From a study of the literature he concludes that hereditary and senile tremors are hardly to be separated, emphasizes the frequent coincidence of tremor with mental feebleness and the signs of degeneration, and quotes Raymond as proposing to group these tremors together, under the name of trembling "neurosis" (*névrose tremulante*).

UN CAS DE MIGRAINE OPHTHALMOPLÉGIQUE.

• Bouchaud (*La Presse Médicale*, April 28th, 1897), stating that ophthalmoplegic migraine is a rare disease, since in a recent analysis of the subject Ballet could cite but 22 cases, reports a case in a woman of sixty-one. The patient, of neurotic heredity, had from twelve to thirty, attacks of ordinary migraine. At the latter age she had severe right occipital neuralgia, and since then has suffered from time to time with vague, diffuse pains in the head. In July, 1895, at the age of sixty, she was seized early one morning with sudden and violent pain in the left side of the head and face, and in the left eye. This lasted all day, and was accompanied by nausea and vomiting. By 8 P.M. she noticed that she saw double, and the pain growing less, was able to sleep. The next morning the pain was gone, but there was ptosis and immobility of the left eyeball. When seen by the author, in January, 1896, there was paralysis of all muscles supplied by the left third nerve. Under electrical treatment the condition is somewhat ameliorated, but a year later loss of power still persists. The author compares his case with the clinical history of ophthalmoplegic migraine, given by Charcot, and while it differs from other reported cases in coming on so late in life, and in permanent paralysis occurring after the first access, he thinks that from its general character it must be considered as an example of this disease. C. L. ALLEN.

ON OEDEMA IN GRAVES' DISEASE.

In the *Edinburgh Medical Journal*, for April, 1897, H. Mackenzie discusses this subject. He divides the oedema of Graves' disease into general and local, pitting and serous, and non-pitting—probably mucoid, and says it may be more or less transitory or permanent. Swelling of the eyelids seems pretty commonly present. It occurred in seven of the ten cases reported in this article. From a study of these ten cases, Mackenzie draws the following conclusions:

"1. A slight œdema, limited to the legs, is not an infrequent accompaniment of the disease. This results from cardiac weakness, and is best met by the administration of cardiac tonics.

"2. A general œdema may be one of the main features of the early stage of the disease, and is not necessarily an unfavorable sign.

"3. General œdema may supervene before death.

"4. Local œdema may affect the eyelids. Either the upper eyelids or the lower may be the seat of swelling, or both may be affected.

"5. Swelling of the eyelids may be an early symptom, or it may come on after some years.

"6. Such swelling is sometimes very persistent, and may remain after all the other symptoms have disappeared.

"7. Sometimes a non-pitting swelling is met with, affecting the lower extremities, resembling the swelling of myxœdema. This swelling, is, however, unaffected by thyroid treatment." ALLEN.

ACUTE ALCOHOL INTOXICATION IN CHILDHOOD. Korn. (Therap. Monatshft, 1897, p. 55.)

Korn reports a case of severe intoxication with alcohol in a boy of nearly seven years, who had taken $\frac{1}{4}$ liter Kümmel whiskey. After initial vomitus, profound coma supervened, which lasted for ten hours, and which the strongest analeptica or cold douches failed to influence. After ten hours, violent tonic and clonic convulsions set in, but the comatose condition still obtained. Tonic spasms of the arms, legs and back muscles strongly simulated tetanus with opisthotonus. Heart action tumultuous, respiration difficult, and only possible with the aid of the auxiliary respiratory muscles. This condition continued throughout the following day, when the breathing and the heart's action became easier. The tetanic condition then gave place to clonic convulsions, but the coma supervened until the fourth day. Temporary lucid intervals occurred upon the following day, but the unconscious condition remained for the major part. During this interval the patient kept striking his arms upon the bed, and seemed to be making attempts to grasp objects, similar to the motions described by Fürstner as excitation of the motor sphere. From the sixth day on symptoms of returning consciousness—hallucinations, illusions—became apparent, followed by rapid amelioration of all signs of meningeal trouble, so that upon the ninth day the little fellow was playing about the house. Such rapid restitution to the normal condition seems frequent after poisoning which has not been acutely fatal. However, in this case, symptoms of an inflammatory meningitis about the motor area recurred after two weeks, and for five days the comatose condition, with convulsions, mostly of the head muscles, again obtained. Finally complete recovery occurred.

Korn compares cases of acute alcohol intoxication in its pathologic-anatomic aspect with the above case, and cites several among published annals, then passing to the treatment, which should be based upon the pathologic changes. Inasmuch as we are almost helpless to restrain the usually very rapid fatty degeneration in the heart and liver tissues in acute alcohol poisoning, our efforts should be mainly directed toward the counteraction of the extreme congestion of the brain and its membranes, and at the same time guard against impending paralysis of the heart and respiration. If the patient is seen early enough, emetics and the stomach pump can be used. Usually, however, vomitus has already occurred, hence the analeptica: strong coffee; liq. ammon. anisat: camphor, etc., are in place. Occasionally artificial respiration must be used. Whenever it can be done, repeated hot baths with cold douches to the head, and afterwards constantly ice to the head and heat to the feet, are indicated. From his experience in

the reported case, Korn advises the careful use of caffeine and anti-pyrin in doses suited to the age of the patient. He calls particular attention to the necessity of extended observation of instances in which a single excessive quantity of that ordinarily evanescent poison, "alcohol," had been taken, and the close study of its effect upon the brain and its membranes, namely marked hyperæmia and long-continued congestion.

STERNE (Indianapolis).

FOUR CASES OF DIPLEGIA IN A FAMILY OF FIVE. D. R. Brower.
(*Medicine*, Jan., 1897.)

The mother of the children whose cases are described is thirty-four, a large, healthy woman, of good family history. Her pelvis is roomy, and her six labors have all been normal. The father is fifty-two, thin and anæmic, has rather a bad heredity and a history of alcoholism and malaria. Syphilis doubtful. He has nystagmus, hesitating speech, some tremor of the hands, no knee-jerks, and some signs of degeneration, as irregularity of the ears, and inequality in the size of the facial bones. Both parents were born in Sweden.

The six children, all boys, were at birth unusually large—weight 14 to 24 pounds—and apparently healthy. The first child, now twelve, never learned to creep, walk, sit alone or feed himself. He could talk a little, until at eight years old he had an attack characterized by fever and delirium, but since then his speech is unintelligible. His memory is good. He has exaggerated reflexes, nystagmus, internal strabismus, athetosis, paralysis of all four extremities, contractures, rigidity and atrophy of muscles. He has no teeth, and there is difficulty in swallowing liquids. His sphincters act normally. Sensation is not impaired. He has never had a convulsion. The facial bones are unequally developed, the ears large and irregular, and the palatine vault high. The mother now thinks it likely that his deficiency was manifest at the age of a month or six weeks.

The second boy died at the age of five and one-half years of some acute disease. He was bright, and presented no evidence of paralysis.

The third boy, now eight, can make no intelligible sounds. He has teeth, but they are notched, and badly developed. He also has paralysis of all four extremities, and other symptoms similar to those of the first boy, as well as similar signs of degeneration. The mother noticed nothing wrong with him until after an attack of erysipelas, at four years of age.

The fourth boy, now six, is fairly healthy, and goes to school, but is not very bright, and has headaches. In the fifth child, now four, the paralysis of the four extremities was noticed two or three months after birth. His symptoms are the same as those in the other cases, except that there is no athetosis or muscular atrophy. The teeth and jaws are abnormal. The sixth child's trouble was noticed soon after birth, and now, at one year old, he presents almost exactly the same symptoms as the last case.

The author is inclined to refer the condition of these children to some prenatal cause. It is not stated whether or no ophthalmoscopic examinations were made, nor is the mental condition of the children fully described.

C. L. ALLEN.

Book Reviews.

A TREATISE ON APPENDICITIS. By John B. Deaver, M.D., Surgeon to the German Hospital, Philadelphia. Containing 32 full-page plates and other illustrations. Philadelphia, P. Blackiston, Son & Co.

While, taken as a whole, this work is an excellent treatise on appendicitis, it is in one sense disappointing, as there is very little in it which is new, and we are forced to conclude that the author's opinion that the disease should invariably be treated by early operation, is somewhat biased when he acknowledges that the 500 cases (personal) which he refers to were all operative cases.

The anatomy of the disease is dealt with in a masterly manner, and is well illustrated. Acute indigestion is given as an important predisposing cause of the disease, and the statement is made that "all cases of appendicitis are directly due to the invasion of certain micro-organisms," while the most important determining cause of an invasion by the micro-organisms is said to be the existing virulence of the colon bacilli.

The "three cardinal symptoms of the disease" are said to be pain, rigidity and tenderness. Vomiting is stated to be common at the onset of an attack, desisting in mild cases and becoming a serious symptom when prolonged. We think, however, that the author makes a rather sweeping statement when he says: "When the three cardinal symptoms are present . . . the diagnosis is unexceptionally warranted," for are not these three symptoms present in a ruptured right pyo-salpinx? We fully agree with him when he says "it is impossible to foretell what will be the outcome of any attack."

While acknowledging that there are a few cases in which he prefers to defer operation, the author says that removal of appendix as soon as diagnosis has been made is the one course to pursue in order to obtain the best results. The "expectant" treatment is summarized as "rest in bed, the judicious administration of laxatives (castor oil or calomel in the early, and salts in the latter stages), restricted diet and the alleviation of pain." For the last he recommends suppositories of asafoetida, and strongly condemns opium.

The preliminary and operative technique as well as the proper after treatment, is minutely described, and the complication and sequelæ which may be looked for are enumerated.

The mechanical make-up of the book is excellent and reflects credit on the publishers, but the reader would have been saved much trouble had the plates been more carefully distributed. GAZZAM.

DIE BEURTHEILUNG DER NERVENERKRANKUNGEN NACH UNFALL (The critical judgment of neuroses following traumatism). By Dr. Alfred Saenger. Published by Ferdinand Enke, Stuttgart. 1896.

In a brochure of ninety pages, Saenger treats of this interesting subject in an effective manner.

The introductory chapter is devoted to an historical view of the subject.

Schultze's remark at the Wiesbaden Conference for internal medicine (1893), that many examinations of workmen with reference to contraction of the visual fields, anæsthesias, etc., were still required, led the author to begin his investigations upon healthy individuals, and such belonging to the laboring classes, who went to the polyclinic for the treatment of various affections as *ulcus cruris*, *gonorrhœa*, etc. None of these cases had ever met with any accident. These individuals, which in all numbered 119, represented all ages and all occupations; a few women were also examined. In each case the sensibility, the reflexes, the vaso-motor phenomena, the special senses and particularly the visual fields, were carefully examined.

Out of the 119 cases, 8 or 6.7% showed, in greater or less degree, a considerable contraction of both visual fields. In 111 cases the visual fields were normal. The author is of the opinion that a concentric though slight contraction of the visual field is a pathological symptom. He insists, however, that this symptom is no pathognomonic sign of the so-called traumatic neurosis. He has found this symptom not only in traumatic hysteria, neurasthenia, hypochondriasis, and other neuroses, but also in alcoholism, in the abuse of tobacco, anæmia, Basedow's disease, and in the early stages of syphilis.

As regards sensory disturbances, Saenger found that 4% of these cases showed a slight degree of anæsthesia, mainly in the form of hypalgesia in the lower extremities. He is inclined to attribute these sensory disturbances to the various injurious exposures incident to the occupations of the examined men.

Exaggeration of the reflex was found so often that the author attributes no pathological significance to it. It cannot be utilized in the judgment of neuroses following injury.

With reference to cardiac action, the pulse was found to be, in a number of cases, slow, in others normal, and in still others, rapid.

Under the title of the ordinary injuries to the nervous system in workmen, the author discusses alcoholism, the abuse of tobacco, syphilis, over-exertion, insufficient nourishment, arteriosclerosis and the hereditary taint.

Many cases are cited, showing that the symptoms which are usually considered of much importance in the diagnosis of the traumatic neuroses, namely, contraction of the visual fields, sensory disturbances, exaggerated reflexes and tachycardia, are often present in individuals who have never suffered from any trauma, but who were the subjects of alcoholism, syphilis, etc.

Are there really any signs pathognomonic of traumatic neuroses? The author says: "On the ground of the published cases and examinations with reference to nervous disturbances in workmen, who have never suffered from any traumatism, it can be strongly maintained that these are no pathognomonic signs for the traumatic neurosis."

Saenger concludes his little book in the following words: "A review of our work shows that the critical judgment of neuroses following traumatism involves one of the most difficult problems to be solved by the physician . . . often for the reason that the condition of the nervous system of the injured individual was not known before the accident." Saenger suggests that every workman before being employed should be submitted to a physical examination with reference to the condition of the visual fields, sensation reflexes, vasomotor state, etc.

The condition of the workingman in Germany seems to be somewhat different from that in this country. It seems that in Germany a laborer who is injured whilst performing his work is entitled by law to receive a certain pension. This doubtless would have a tendency

to engender and to prolong traumatic neuroses. The result of the investigations of Saenger, at least with reference to German laborers, are not without importance. It is a pity that the same investigations did not take in more women of the lower classes, as well as men and women belonging to the upper ranks. As syphilis, alcoholism, the abuse of tobacco, arterio-sclerosis, hereditary taints, etc., are as common in the upper walks of life as in the lower, it may be presumed that the results of the examination of individuals pertaining to the upper classes would be analogous to those obtained by Saenger. If that be the case it must follow that a rational judgment of the extent of dependence of a neurosis upon a trauma, would be no easy matter.

MEIROWITZ.

A SYSTEM OF PRACTICAL MEDICINE BY AMERICAN AUTHORS. Edited by Alfred Lee Loomis, M.D., L.L.D., and William Gilman Thompson, M.D. Volume I, Infectious Diseases. Lea Brothers & Co., New York and Philadelphia.

The first volume of the System of Practical Medicine, edited by Loomis and Thompson, is devoted to the infectious diseases. A glance at the list of distinguished authors is sufficient to satisfy any one that the subjects have been handled by men who are recognized authorities in their special fields. It is impossible to do justice in a brief review to a work of this character, and it is to be regretted that the theme is one which renders a lengthy abstract somewhat unsuitable for a neurological journal. The book has been published in the manner in which American works are usually put upon the market. The typography and illustrations are excellent.

SPILLER.

JOURNAL DE NEUROLOGIE ET D'HYPNOLOGIE. Edited in Brussels.

This newly-founded journal which appears twice a month is under the direction of M. X. Francotte, Professor of the Neurological and Psychiatric Clinic at the University of Liège. It is edited by Dr. T. B. Crocq (chief editor) and by Dr. Mahaim and Dr. Swolfs, with the collaboration of many prominent neurologists.

In making the announcement of the journal, the editors express the hope to make it indispensable to all the physicians who wish to be *au courant* of neurological and hypnological science. Judging from the names on the list of collaborators, it may at any rate be expected that the journal will obtain prominence, and in looking over the numbers published so far, we strike many articles which attract our liveliest interest, among others those of Van Gehuchten (Contribution à l'étude du faisceau pyramidal) and of de Potter (Etude sur l'hypnotisme).

The periscope department seems excellently managed. The list of abstracts is large, comprising not only neurology and hypnology, but also psychiatry, electrotherapeutics and anthropology.

Besides a good bibliography we find complete reports of those society proceedings which treat on neurological or allied subjects.

ONUF.

THE
Journal
OF
Nervous and Mental Disease

AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-third Annual Meeting, held at St. John's Parish Hall,
Washington, D.C., May 4th, 5th, and 6th, 1897.*

The President, Dr. M. A. Starr, in the chair.

A CASE OF SYRINGOMYELIA WITH TRUNK
ANESTHESIA.

By HUGH T. PATRICK, M.D.,

Professor of Neurology in the Chicago Policlinic; Assistant Professor of Nervous
Diseases, Northwestern University Medical School; Consulting Neuro-
logist to the Illinois Eastern Hospital for the Insane, etc.

Mr. N., aged forty-four, of good habits and negative family history, was sent to me January 16th, 1897, when the following notes were taken. With the exception of slight chronic rheumatism, which has affected his left leg on and off for about twenty years, but which never disabled him, he was perfectly well until ten years ago. While he was at work, a heavy weight came down upon his shoulders, bending him forward and straining his back, and at the same time he was pressed in the abdomen by some implement. The injury was not very severe, as he resumed work in a day or two, but his back was troublesome for some time afterward, and it has not been as strong since. About six months after this accident he began to be aware of some clumsiness and weakness of the legs, and noticed

that they tired more easily than before. This disability gradually increased, and about a year ago the hands began to show some weakness, which has also grown more pronounced, although he has been able to continue his work of repairing cars. Sensory symptoms have been wanting, except discomfort in the back, sometimes amounting to

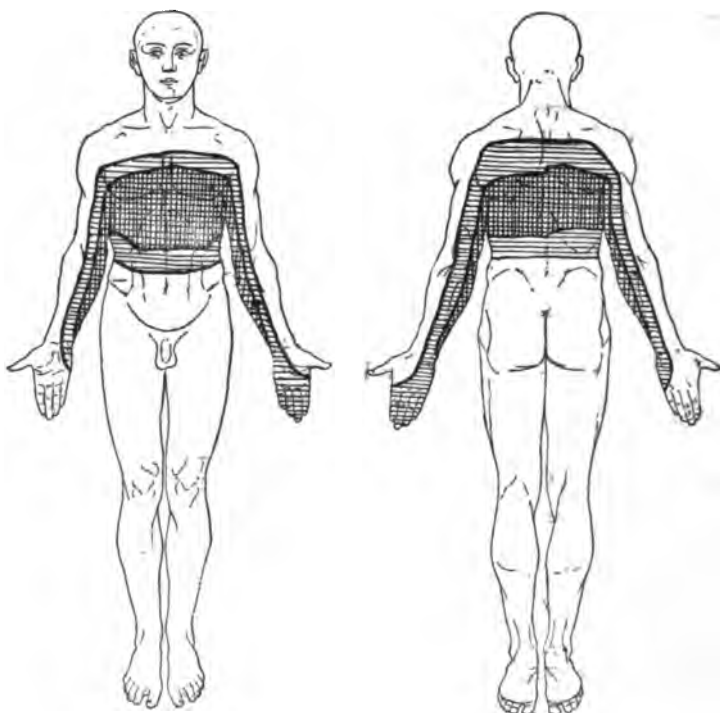


FIG. I.

FIG. II.

SYRINGOMYELIA.

Perpendicular shading—anesthetic areas.

Horizontal shading—analgesic areas.

pain. He now has considerable difficulty in walking. The gait is typically spastic, with some uncertainty, and the muscles of the lower extremities are slightly paretic. The arms are reasonably strong in proportion to the muscular development, but the grasp is below normal and the inter-ossei are decidedly weak, more so in the left hand. There

is beginning atrophy in the small muscles of both hands, more distinct on the left side, and these muscles show partial reaction of degeneration. No atrophy is discoverable elsewhere, except high up in the back, where partial reaction of degeneration is also found. In one of these muscles, the supraspinatus, there is the myotatic contrac-



FIG. III.

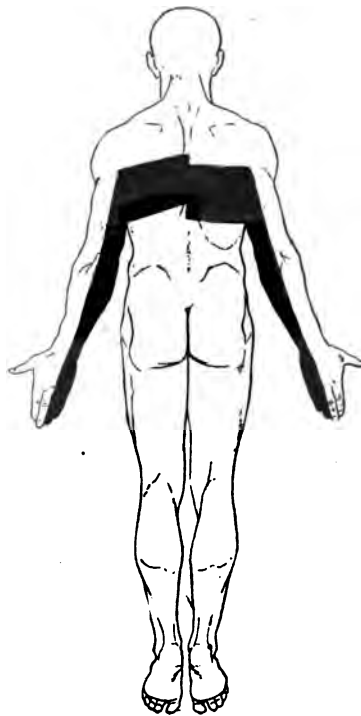
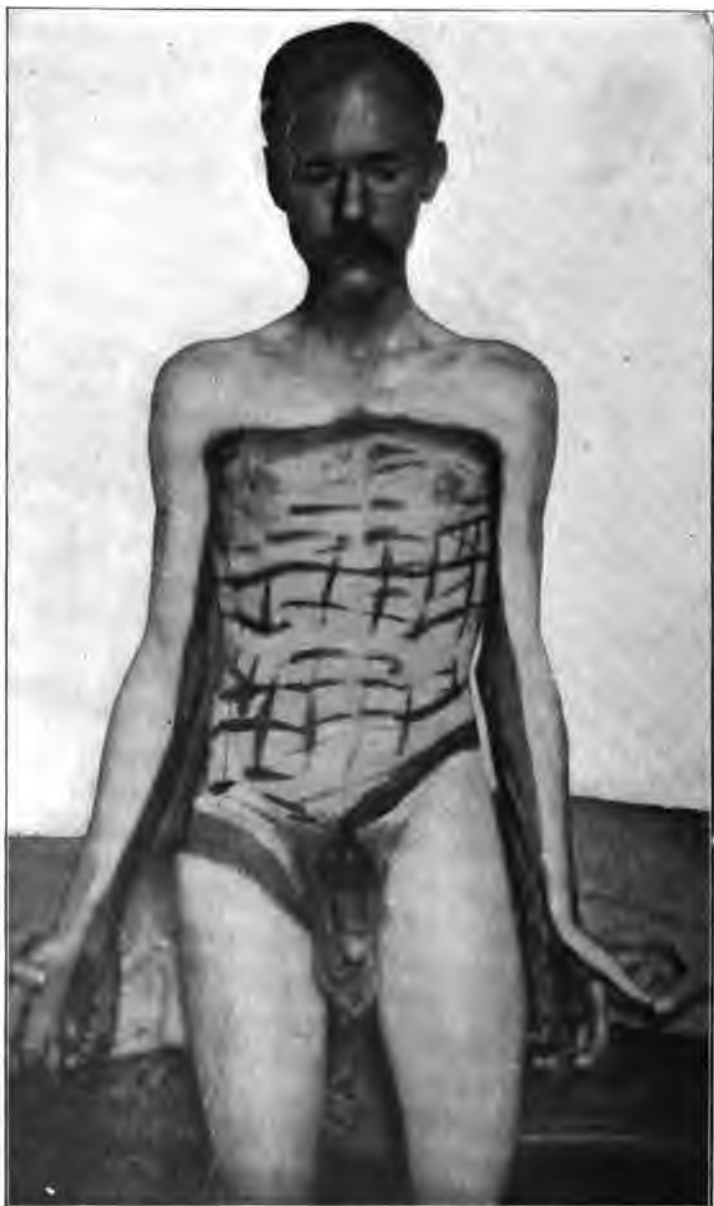


FIG. IV.

TABES.

Shading indicates the anesthetic areas. Analgesia was limited to a very narrow zone along the middle of the anesthetic areas.

tion that is almost exclusively found in Thomsen's disease. That is, after momentary stimulation the muscular contraction persists for several seconds, causing an almost tumor-like swelling of the affected muscle. So far as I know, this has never been described as occurring in syr-



TABES.

Stained surface indicates area of tactile anesthesia. Analgesia was limited to a much narrower zone.



TABES.

Stained surface indicates area of tactile anesthesia. Analgesia was limited to a much narrower zone.

ingomyelia. The knee jerks are greatly exaggerated and foot clonus is well marked. The left wrist tap is scarcely obtainable, the right is very distinct. The cranial nerves and visual fields are normal.

It is, however, the sensory symptoms of the patient that seem to me to be of particular interest. Indeed, it is for them alone that I report the case. The tactile sense is normal, except for a band of anesthesia about the trunk, extending in front from the third intercostal space to about four inches above the umbilicus, and behind from the third dorsal spine to the interval between the tenth and eleventh spines. The zone is rather wider in front than behind, and slightly lower on the left side. This anesthetic band extends onto the inner surface of the arms, embracing, perhaps, one-third of the circumference above, and tapering downwards to gradually disappear, somewhere between the middle of the forearm and the wrist. It is impossible to define the borders exactly. There is loss of pain and temperature sense in an area that covers the fore-mentioned anesthetic region and overlaps it by about four inches on the trunk and one or two inches on the arms; extends below the wrist on the right side, and involves most of the hand on the left. (Figures I. and II.) There is also some diminution of the temperature sense over the left hip and thigh, but it is so very slight in degree and uncertain in extent that no distinct area could be outlined.

In a short paper on trunk anesthesia in tabes² I surmised the possibility of a similar condition occurring in syringomyelia, and, indeed, Laehr³ has described it, more or less typical, in seven cases of this disease. None of them, however, showed it in such a striking way as does this one of mine.

The symptom seems to me to be very suggestive. In tabes we have a lesion that causes anesthesia and anal-

²New York Medical Journal, Feb. 6th, 1897.

³Arch. f. Psych. u. Nerv., 1896. Band XXVIII., Heft 3.

gesia corresponding to spinal segments (Figures III. to VI.), the anesthesia being much greater in area than the analgesia. In syringomyelia exactly the same distribution may be found, but reversed qualitatively; that is, the analgesia is more extensive than the tactile anesthesia.

DISCUSSION.

Dr. Sachs—I was intensely interested in Dr. Knapp's paper, and I think we are all under obligations to him for bringing the matter before us in such a lucid way. I have been struck within the past year by a definite group of cases which were distinctly of spinal origin, in which there was a considerable amount of anesthesia; and I have also been struck by the fact that in these cases the anesthesia was not of the spinal type, while every other symptom was entirely in keeping with the diagnosis of spinal disease. I think on a first and hasty examination these cases might have been diagnosticated as syringomyelia, but I have very good reason to believe they are cases of syphilitic infection of the cervical portion of the cord. I have been particularly interested in the explanation which Dr. Knapp gave, because if this peculiar and idiopathic form is to be attributed rather to interference with the roots than to interference with the spinal cord, it would fall in much more readily with the supposition of a syphilitic process than with a supposition of syringomyelia. I believe there are cases of syphilis of the cervical portion of the cord. This special form of anesthesia has been spoken of as cerebral anesthesia, but I think, perhaps for the present, a better description for it would be a regional anesthesia, rather than cerebral.

The President—I have recently been over a large number of cases which have been published within the last four years on the subject of anesthetics in the body with definite lesions in the cord. I would call attention to the paper by Kocher, of Berne, describing seventy cases of localized injuries to the spinal cord, occurring in his practice, from fractures and dislocations; a paper of the greatest value,¹ and accompanied also by a diagram of the surface of the body, and with an attempt to limit the distribution of anesthesia in lesions of the various segments.

It seems to me difficult to lay down a definite diagram of the body as related to the areas of the cord. My work, four years ago, was only partially successful. The results of Head and of Kocher have been somewhat different from mine. It is a matter which requires further study, and I think that individ-

¹Mittheilungen aus den Grenzgebieten der Med. u. Chirurgie I. 4.

ual variations must be admitted, just as they have to be admitted in regard to the distribution of sensory nerves in the skin. At the same time it is well worth while to record these cases, because lesions in the cord are not absolutely level in their distribution, but are irregular in their distribution, and hence each case may differ from others. I think Dr. Knapp's diagrams are very ingenious ones.

Dr. Knapp—Of course, the ordinary term which has been used for these anesthetics, where the boundary is distinctively at right angles to the axis of the limb, is segmental, but that term is open to the objection that it might be referred to lesions of segments of the cord. Inasmuch as it is rather characteristic of some hysterical anesthetics, I applied the term cerebral to it, without meaning to imply anything in regard to the real origin of the trouble.

The diagram I claim no credit for; it is by Ballet.

We certainly see clinical variations in cases where the anesthesia is of the spinal type, and part of these variations may be due to the variations of anatomical distribution in individuals. In one case the first dorsal segment may supply the skin of the ulnar border of the forearm only, in another case it may also supply the skin on the ulnar border of the hand as well. But where, on the one hand, we see anesthesia that passes in ribbon-like strips down the hand and involves, perhaps, the thumb and the radial border of the hand in one case, and in another case the ulnar border of the hand and little finger, these anesthetics are clearly distinct in their type from those that stop short at the wrist like a glove. The origin of the latter seems to me still obscure, and Ballet's diagram explains only a part of them. In cases of tabes the diagram does not seem to be wholly satisfactory.

Dr. Theodore Diller read by title the following papers:

A CASE OF SYRINGOMYELIA,

A CASE OF ACUTE SOFTENING OF THE PONS.

Dr. F. X. Dercum read by title:

STUDIES IN SCLERODERMA.

A CASE OF SO-CALLED POLIENCEPHALITIS.

A PRELIMINARY NOTE.

By HUGH T. PATRICK, M.D.

Professor of Neurology in the Chicago Policlinic; Assistant Professor of Neurology in the Northwestern University Medical School; Neurologist to the German Hospital; Consulting Neurologist to the Illinois Eastern Hospital for the Insane, etc.

A lady, aged thirty, of negative family and personal history, was first seen September 30th, 1896. The first symptom of her existing illness had appeared about two months before as a well-marked bulimia which remained the sole manifestation for two or three weeks, and gradually diminished as the other symptoms appeared. The next trouble noticed was difficulty in swallowing, and to this were added confused vision, then distinct diplopia, a feeling of thickness and clumsiness of the tongue, and general weakness with special involvement of the shoulder and upper arm muscles, the grasp remaining comparatively strong. There had been no distinct cephalalgia and no mental disturbance.

Examination showed external ophthalmoplegia, incomplete bulbar paralysis, weakness of the neck muscles, almost complete paralysis of the deltoids and general myasthenia. Lateral movement of the eyes was almost *nil*, vertical motion was better but very limited; there was no distinct ptosis, the pupils reacted to light but not to accommodation. The lower facial and neck muscles seemed weak, but there was no localized paralysis or atrophy, excepting, possibly, some wasting of the sternal head of the right sterno-mastoid. The tongue was weak and seemed small, but was not atrophic and was fairly well protruded. Deglutition was difficult, but there was no regurgitation through the nose. Although the deltoids were powerless, they were not atrophied, and they reacted promptly to the faradic current, as did also the muscles of the face and neck.

The lower extremities were weak only, and the knee-jerks were normal.

For a few days after this she was thought to improve somewhat, but soon came to a standstill, and October 10th, that is, ten days from the time I saw her, she grew decidedly worse, was greatly troubled by the accumulation of mucus in the throat (and probably trachea), and by increased difficulty in swallowing. Two days later she was still worse, respiration became greatly embarrassed, the general prostration extreme, and the temperature, before normal, rapidly rose to 102. I was hurriedly sent for and arrived a very few minutes before death. I found her perfectly conscious but laboriously gasping for breath. There was no stenosis, the respirations were rather slow and quite shallow, the mouth was open, with each inspiration it was still further opened, and every particle of vitality seemed concentrated in the effort to accomplish the respiratory act. The diaphragm was apparently paralyzed, but the neck muscles acted. The pulse was rapid, but not so weak as the patient's state would indicate. On demand she protruded the tongue, pursed the lips, grasped my hand and moved the lower extremities. A moment later the gasps became less frequent—exactly like those of a dying person, the eyes closed, the pulse flagged, and she apparently became unconscious. In about half a minute, however, respiration improved, the pulse came up and she opened her eyes. This really was only momentary, and she died about five minutes later, respiration ceasing before the pulse.

At the autopsy, which I made ten hours later, nothing abnormal could be seen. The cord was not obtained. The brain, pons and medulla were hardened in Müller's fluid, and sections were stained with uranium carmine, hematoxylin-eosin, according to Weigert, Pal, Rosin, and Nissl, excepting, of course, the alcoholic hardening required by the latter stain. Unstained sections were mounted in glycerine, and at several levels slices were stained according to Marchi.

The microscopic findings are unexpectedly meagre. There is no increase of nuclei, no migration of leucocytes, and, so far as I have examined (about one thousand sections), no hemorrhages, except one very small one. There is no degeneration of fibres excepting, possibly, a few in the intramedullary root of the facial. The capillaries and arterioles are enormously distended throughout, and in some places the perivascular spaces seem to be also distended. Some of the cells in the facial and abducens nuclei seem to show, with the Marchi stain, evidence of slight degeneration. A large number of granules, chiefly grouped around the nucleus of the cell, are stained an opaque black. Some such fine, black masses are often found in normal cells stained with osmic acid, but not to the extent seen in these specimens. Abnormal changes in the other nerve nuclei are almost problematical but, I think, exist.

From the clinical picture and microscopic examination, as far as made, the case seems to me to be one of toxemia of some kind, and a transition case between two classes of nervous disease heretofore described as entirely distinct. On the one hand, the acute polienccephalitis of Wernicke, acute poliomyelitis and possibly acute neuritis, and, on the other hand, the cases of ophthalmoplegia and bulbar paralysis that have now and again been reported with negative microscopic findings, and the clinically observed cases of supposed purely functional character—so-called asthenic bulbar paralysis. My case indicates that all will be found to belong to the same class.

In the light of modern therapeutics, I must venture to suggest that the proper treatment of such cases would be the procedure rather picturesquely termed "washing the blood"; that is, copious phlebotomy with simultaneous transfusion or subcutaneous injection of normal salt solution.

Session of May 5th.

A STUDY OF A CASE OF ENCEPHALITIS, WITH CHANGES
IN THE PIA.

Dr. Alfred Wiener, of New York, read a paper on this subject, and reported the case of a child, three years of age, who, after meeting with an accident, suffered from an attack of acute (non-suppurative) meningo-encephalitis. The child recovered after six months of illness. It remained perfectly well, and after an interval of three years, just subsequent to an attendance of three months at school, had a second attack which proved fatal. The autopsy and microscopic examination showed an involvement of the pia as well as of the brain, and the characteristic picture of acute non-suppurative hemorrhagic encephalitis. He believed the first attack was due to traumatic influences. The second attack was supposed either to be due to influenza, on account of the suspicious catarrhal symptoms present at the beginning of the attack, or to be a result of mental strain in a child predisposed to such an attack. Special attention was called to the involvement of the pia, which represented a pathologic condition often found in simple fibrinous pleurisy.

Dr. Joseph Collins reported, as a contribution to the symptomatology and pathologic anatomy of

ACUTE, HEMORRHAGIC, NON-PURULENT ENCEPHALITIS.

the history and autopsy record of the following case:—

A young man, twenty-two years old, was admitted to the City Hospital in the latter part of January, of the present year. The history that was obtained from his parents was, that he had never been sick, but that he had always been somewhat delicate and never as bright mentally as the average lad. His mother is positive that he was quite well until the present illness, which came upon him very suddenly while he was repairing a door latch. He suddenly fell over, without making any outcry, and since this event has not spoken a word. This was two weeks before he

came into the hospital. Very little could be learned of the boy's condition during that fortnight, as he did not seem to have had any regular medical attendance. The mother says that he was "out of his head" most of the time, restless and continually moving even while asleep; that he refused to take food, soiled the bed and did not recognize anyone about him. He was carried to the hospital on a stretcher and put in the wards of Dr. Brewer, who requested me to see him.

The patient was in a dazed, staring, non-observant condition, with the neck slightly arched backward and stiffened. The continual movement of the patient impressed one greatly on seeing him for the first time. The movements, on analysis, were seen to affect particularly the left hand and forearm, and were almost exactly like those described as athetoid. They were rhythmical, continuous, and consisted of repeated flexion, extension and rotation of the hand, and involved the entire upper extremity. The entire left side of the body, including the face, was spastic. There was no movement of the right side of the body, and this side, instead of being spastic, was flaccid to such a degree that one was inclined to believe that there was a slight hemiparesis, but this side of the body could still be moved. There were peculiar in-coordinated movements of the eyeballs when they were directed to the extreme right or left. The sensorium was very much obtunded, but there was no appearance of coma. The patient made no response to questions, and in no way gave evidence that he understood what was said to him, nor did he take particular notice of members of his family who visited him. The pupillary light reflex was present, but the pupils did not contract to a very small point. There were no cranial nerve palsies. He neither rested nor slept. His general condition was aptly and fully described by the term *typhoidal*; the general aspect was one of a patient suffering from some profoundly infectious disease. He emaciated rapidly, so that after a few days in the hospital nearly all the adipose tissue had disappeared. The temperature was constantly slightly elevated; the pulse was sometimes about normal, sometimes beating as often as one hundred times per minute; the respirations were slow.

The patient's condition became every day more un-

satisfactory, the sensorium more sluggish, the athetoid movements of the left hand less constant and pronounced, the rigidity more marked in the left leg, but less so in the upper extremity; and the flaccidity and paresis of the right side more strongly in contrast. There was incontinence of urine and feces, and a bed sore developed over the sacrum. There was conjugate deviation of the eyeballs toward the left, and Cheyne-Stokes respiration preceding the *exitus letalis*.

On opening the calvarium the dura was found to be normal. There was marked excess of the cerebrospinal fluid, which was of its customary appearance. The pia was deeply congested and somewhat edematous, especially over the central regions. The supertemporal lobe of the left hemisphere, and the lower third of the Rolandic area of the same side, appeared to be edematous, and had a pulpy, soggy feel, which was in marked contrast to the other convolutions of the brain. On the right hemisphere there was a depression situated at the junction of the Sylvian and central fissures, about an inch and a half in diameter, which was softened, pultaceous, and contained a serous, sanguinolent fluid. This area of softening involved principally the anterior central convolution.

If we recapitulate the symptoms in this case, they are briefly:

1. Sudden, unheralded onset, apoplectiform in character.
2. Somnolence; obscuration of the sensorium; sensory aphasia.
3. Spasticity of the left side of the body; athetoid movements of the left hand.
4. Flaccidity of the right side of the body; right-sided hemiparesis.
5. Incoordination of the eyeballs on lateral movement.
6. Stiffness of the neck and great restlessness.
7. General accompaniments of profound infection; typhoid state.
8. Conjugate deviation of the eyeballs; Cheyne-Stokes respiration.

All these symptoms were dependent upon an acute, non-purulent encephalitis with two principal foci, one of which was in the left hemisphere and involved the supertemporal convolution—thereby causing aphasia—and the

lower part of the motor area. The focus in the latter region was in a more advanced stage of disintegration than those elsewhere in the brain, and to destruction of tissue by this portion of the focus were due the flaccidity and right hemiparesis. The other focus was in the lower part of the motor area of the right hemisphere, and caused the athetosis and rigidity; but as it became more disintegrated as the disease advanced, the phenomena of irritation ceased and paralytic ones were manifested. A consideration of these symptoms and of the morbid findings seems to me most instructive and contributory to our knowledge of acute infectious encephalitis.

Dr. Spiller.—It seems to me that the resemblance of this cortical form of inflammation to the poli-encephalitis superior acuta of Wernicke, of which Dr. Wiener has spoken, is striking, but I should go a little further than he has gone. The poliomyelitis anterior acuta has pathologically much in common with the two forms just mentioned. I have been able to examine specimens from acute encephalitis and from acute poliomyelitis, and I cannot say that there were any essential differences in the nature of the two processes. In both the small vessels were much distended and capillary hemorrhages were easily seen. The case of acute poliomyelitis to which I refer has been reported by Redlich, and he has stated that the foci of inflammation were not limited to the cord.

I may call the attention of the Association to the case of encephalitis reported last year by Lloyd and Sailer. In that case also the foci were widely distributed. The patient was supposed to be suffering from typhoid fever until paralysis of ocular muscles made the diagnosis improbable.

I saw a case with Dr. Mills some time ago which presented many of the symptoms of brain tumor. Dr. Sailer, who performed the necropsy, told me that he had observed multiple foci, which appeared to be similar to those reported by Dr. Lloyd and himself.

Dr. Dercum, of Philadelphia.—I would like to ask whether there were evidences of infection found in the general autopsy; what, for instance, was the condition of the kidneys, whether infarcts were present, or any other signs which would suggest an infectious disease.

Dr. Wiener.—There was an acute congestion found in the kidneys.

Dr. Sachs.—I would refer to a practical difficulty I experienced in the first case mentioned by Dr. Wiener, and one or two others; the practical difficulty of differentiating in the earlier stages between cases of meningitis and these cases of en-

cephalitis. Even though Dr. Wiener found distinct evidences of involvement of the pia in this case after the first attack, it is of course doubtful whether that involvement existed at the first attack. This differential diagnosis is the great stumbling block in the way of making a diagnosis of encephalitis in the first beginnings of the disease. At present we have, so far as I can see, no point of diagnosis, except the general gravity of the disease and the fact that a large majority of these cases of encephalitis do not take the rapidly fatal course that the cases of meningitis do. The chronic forms of encephalitis, the combination of bulbar encephalitis with myelitis, can be understood much more clearly than they formerly were. The entire question of encephalitis is a burning one, and I think we must not only try to clear up the question of the pathology of the disease, but we also need a great deal more study with reference to the differential diagnosis between meningitis and encephalitis. In some, if not a majority of the cases, an encephalitis may be combined with a meningitis.

Dr. J. J. Putnam.—I did not hear Dr. Wiener's paper, but it seems to me that we do not, perhaps, sufficiently realize to what an extent the symptoms are of toxic origin in these cases, and this seems the more probable when it is remembered that the outcome is so often relatively favorable. Even where meningitis is present the actual influence at work, or a part of it, may be an accumulation of toxic substances.

Dr. Herter.—I remember a very instructive case, in which the microscopic examination of the patient (a child) showed that upon the right side there was a considerable area in which the pia had undergone a productive inflammation. There was no evidence of degeneration in the cortex.

Dr. Wiener.—I had hoped some of the members might have had some explanation to offer in regard to the inflammation of the pia, which I found in this case. I found such an extensive involvement that I thought many of the symptoms were due to this inflammation, viz., that they were symptoms of irritation.

I agree with Dr. Putnam that the violence of the symptoms is due, undoubtedly, to the toxic substances which might be present in the blood in these cases.

Dr. Joseph Collins, of New York, and Dr. B. Onuf, of New York, read by title the following paper:

THE PATHOLOGICAL ANATOMY OF HUNTINGTON'S
CHOREA.

THE NATURE AND TREATMENT OF SPASMODIC TORTICOLLIS.

Dr. G. L. Walton, of Boston, read this paper. He considered spasmodic torticollis an affection of the cortical centres for rotation of the head. The pathogeny is not settled. The fact that it is sometimes easily inhibited does not establish a mental origin. Gross organic lesion is not present. Long continued habit may merge into spasm, as seen in certain occupations. Eye strain sometimes plays a part through causing faulty posture (oblique astigmatism, muscular insufficiencies). In one case it followed the wearing of a glass, which increased instead of relieving an oblique astigmatism.

The course of the disease is progressive.

The principal muscles affected are the sternomastoid, splenius capitis, complexus, trachelomastoid, and the inferior oblique.

The most common form implicates the sternomastoid of one side and the posterior rotators of the other; less frequently the spasm is limited to the sternomastoid, occasionally to the posterior rotators of both sides (retrocollis), and rarely to the sternomastoid and posterior rotators of the same side.

Treatment other than operation is ineffectual in well-established cases. Simple nerve section and nerve stretching are unavailing. The only operations to be considered are resection of nerves and section of muscles. Operations are generally too limited rather than too extensive. In most cases it will be necessary to resect the spinal accessory and the first three posterior branches of the cervical nerves. It will generally be wise to cut also the affected muscles. Muscle section alone has given good results (Kocher), but there is no reason to abandon nerve section.

Absolute cure cannot be expected in over half the cases, improvement occurs in a great proportion, and failure in a certain proportion.

Dr. Dercum, of Philadelphia.—We are indebted to Dr. Walton for his very able summary of the present status of the subject. I think, however, that we ought to be a little conservative with regard to our view of the pathology of the affection, especially that the lesion is limited to the cortex. I do not feel inclined to so regard it. It seems to me that the simple fact which was mentioned by Dr. Walton, that we sometimes have the disease coming on in the course of an occupation (I have seen it come on in quite a number of weavers) so that it

simulates an occupation neurosis, would favor the idea that we have to deal with a trouble not only cortical but rather with a *corticospinal* disturbance of function.

With regard to the measures proposed for relief in these cases, I have, I think, never seen a single instance of absolute cure. I remember very well a case that was operated on at various times for me by Dr. Keen, a case in which at one time the spinal accessory nerve was resected, at another the insertion of the sternomastoid was cut, and still later the nerve supply of the short rotators was resected; and yet, notwithstanding the very great improvement in that case, there was subsequently some return of the movement. It seems to me that the muscles that may take part in the rotation of the head are so numerous—there are some nine or ten, possibly eleven, that may be involved—as to make the task of paralyzing all these muscles almost hopeless.

In regard to the benefit obtained in some of these cases by means of the rest method, I do not entirely agree with Dr. Walton. I believe that some of these cases are permanently benefited by prolonged rest, especially if they are made to lie continuously on a low pillow, are fed by the nurse and not even permitted to leave the bed to void the bladder or bowels; in other words when a rest almost absolute is instituted.

Again, there is one drug that does do some good in these cases, and that is gelsemium, which was introduced for the treatment of these cases some years ago by Weir Mitchell. Used in fifteen drop doses, three, four or five times a day, this drug undoubtedly does decrease the movement and gives a relative degree of comfort to the patient.

My own habit is to advise surgical interference in the majority of cases, and I think it better to divide the operation into two different periods, though I always defer to the surgeon on a point like this. I have never seen much benefit following section of the spinal accessory alone.

With regard to surgical interference, I think we ought to look at the matter from a philosophical standpoint, and we ought to expect as little from it, unless the operation be extensive, as we should expect in operating for writer's cramp, because there is a certain analogy between the two diseases.

The points to which Dr. Walton has called attention, the refractive troubles and color deficiencies in the eye, are of great interest, and I agree with him that we have to some extent neglected their study.

Dr. Dana.—There are, in my experience, three different phases which spasmodic torticollis assumes. One is the common form, seen usually in rather early life, and apt to be associated with neurasthenia and some hysterical manifestations. It is often the sequela of some shock or exhausting work, and is, as has been stated, allied to the professional neuroses. A

second class of cases, somewhat similar but rather more serious, occurs later in life; it starts in like the facial tic and the facial neuralgias, and is a degenerative neurosis. The third, and by far the most serious kind, is one which develops at the choreic age, between twelve and fifteen, and has at first choreic symptoms associated with it. The malady steadily progresses, and the spasmodic movements of the neck associated with choreic movements finally become very severe. Such cases are progressive and incurable. In the treatment of torticollis, it is really only in the first class I have mentioned that we expect to benefit very much.

In regard to its pathology, I quite agree with the views expressed so positively by Dr. Walton. It seems to me that the most rational view of most cases is to suppose that the malady lies in the cortex of the brain. In one patient who suffered from that form of torticollis which I have described as the third type, and who had very severe spasmodic manifestations, death ensued, and I found a chronic leptomeningitis of the convexity of the brain, with some degeneration of the cells beneath.

As for the treatment of this trouble, I can quite agree with Dr. Dercum as to the value of rest; in fact, I do not think there is anything that is much good except rest, and I believe that if this is carried out really persistently, to an extent, perhaps, that few patients are willing to submit to, it will cure some cases and relieve a great many others. This is particularly true if you can take hold of the case in its early stages, a year or two after it begins. I think, too, that careful massage by intelligent persons helps a great deal in the management of the disease in connection with the rest, and sometimes even massage alone, in the early stages, carefully applied, will do a great deal of good, more than any other single measure, to those persons who will not take rest.

I do not believe myself that there is any drug that really does much good. I have tried gelsemium, and tried everything, and have never been able to convince myself that any drug did any permanent good, and I have not seen any good results from the opium treatment which Gowers recommended. It is always dangerous to recommend this.

As to the surgical treatment, it seems to me theoretically altogether irrational; practically I have never seen it, or any kind of mechanical treatment, do any good whatever. At the same time, I should be willing to admit that I have not had cases treated by the combined muscle section and nerve section, and I think that this extremely radical measure, where you practically cut off the head for a time, may prove a sort of a supplemental rest treatment, and in that way be classed under the head of rational measures.

Dr. Charles K. Mills.—Although I have had abundant

clinical experience with this affection, having seen quite a large number of cases, and having treated them by all sorts of methods, I do not find that I have much to add to what has been said here this morning.

With regard to treatment, one form of treatment has seemed almost as good as another, but I would refer to one method of treatment which gave me my only real success in this disease. It was employed on a patient whose history was reported a number of years ago, and who remains to this day well, after having had a most serious case of torticollis. This patient got well under the use of the actual cautery, frequently repeated, two or three scores of times altogether, and the use of the iodide and the bromide of potassium.

I have treated cases with various sorts of drugs, and have had the most result from the use of gelsemium. In one case in particular the patient was very much relieved for a considerable time. The drug was pushed to toxic doses, fifteen or eighteen minims were administered every three hours toward the end of the treatment, and when we had thoroughly poisoned her with the drug the movement ceased and she remained better for some time, but the attacks afterwards returned.

As to the surgical treatment, I have had some experience. In a given case, the spinal accessory nerve was stretched, and then the nerve was tied with silver wire after the manner recommended by a London surgeon, and then the nerve was resected. I have seen several cases of muscle operation, and have never seen any decided or persistent results from any operation. Personally, I would confine the surgical and medicinal treatment to the following: The administration internally of gelsemium; the use in some cases of the iodides; the actual cautery applied to the back and the sides of the neck; and nerve stretching. I believe that you will get as good results after all from nerve stretching in these cases as you will from the more serious operations.

With regard to the nature of this affection, I think we are all "at sea." There does seem to be some reason for believing that some of the cases are of cortical origin. I am not, however, inclined to accept this explanation for a majority of the cases of the special type to which, I think, we all allude in this discussion.

The affection seems to me similar to some of the disorders of ocular movement. We have here probably an affection in which we have a disturbance of the cervical co-ordination.

Dr. M. H. Richardson.—The surgical treatment of spasmodic wry neck has been a subject of great interest to me for several years. In an encouraging percentage of cases great improvement has followed operative treatment, greater than

has followed medical or palliative measures. Indeed, in practically all my cases operation has been undertaken only after the failure of all other methods. Although my experience has been almost entirely in favor of the surgical rather than of the medical treatment, I do not feel justified in operating unless the neurologist has declared himself satisfied that surgery offers the only reasonable probability of relief.

With reference to the pathology of spasmodic torticollis, I have observed that the nerves supplying the affected muscles seem healthy; they are large, strong bundles, unchanged in gross appearance; they respond quickly to mechanical irritation. In obstinate neuralgias, on the other hand, trunks like the third division of the fifth are evidently diseased. I have seen cases in which the affected branch was soft and friable, breaking under the slightest touch, and making avulsion impossible.

The affected muscles in spasmodic torticollis present no abnormal appearances.

These facts, with others, especially that of the rapid shifting of the spasm to remote groups, seem to point to a central rather than a peripheral origin.

The results after all methods of treatment present features both of encouragement and of discouragement. No method is unworthy of trial, and none is always successful. Every means should be employed which has been found after faithful use to be of value. Not that it is justifiable to waste time in the use of doubtful methods in cases of extreme severity, for it is sometimes clear that nothing short of the most extensive dissection offers a reasonable chance of benefit.

In moderate cases—cases in which the spasm is limited, the pain slight, the disability bearable—massage, electricity, enforced rest, should be faithfully and persistently employed. In cases of great severity, on the other hand, I am fully convinced that extensive destruction of the nerve supply, with perhaps muscle division, should be resorted to early rather than late, because of the danger of extension of the spasm to remote groups, and of the deeper fixation of the cause, whatever it may be.

Dr. W. W. Keen.—I was much interested in Dr. Dana's remarks. I was not aware that any necropsies had been made to determine whether the cases were cortical or not. I do not know whether there was any microscopic examination made in Dr. Dana's case or not; that, of course, would be very important. [Dr. Dana said they were made.] I have had one patient who suffered so severely that I proposed to him a cortical operation, having excised both the spinal accessory and also the posterior cervical nerves, but he never quite reached a favorable decision, and I was not sure enough of my ground

to urge it; besides that, the determining factor was that he finally became so much better that he was able to return to his occupation, and therefore was not willing to have a further, and possibly rather dangerous, operation attempted.

It seems to me that in the matter of treatment we ought to be very sure: first, that the medical means that have been alluded to have been employed, and particularly gelsemium and absolute rest—the rest that was spoken of, I think, by Dr. Dercum—the patient not being allowed to move the head in any way. We ought to take a hint here from the value and success of this treatment in writer's cramp and such allied occupation spasms. The rest that we are able to secure in these cases should be as absolute as possible. Whether it is necessary to place a helmet or other apparatus on the head of the patient, in order to secure this absolute rest, I do not know. Then also, the eye condition should be looked into very carefully. But the moment that these means have been tried, and tried faithfully, and have failed, then I think the earlier the operation is done the better. We all know that in spasmodic affections, for example spasmodic neuralgia, there is a habit set up; a habit of pain as well as a habit of muscular spasm, and to delay operation is dangerous.

As to the types of torticollis, I have seen it in very many cases of hysterical persons, of course, but I have seen it in others, notably in the person to whom I referred a moment ago, who was a man very much of the build, vigor and robustness of Dr. Richardson himself, and as far as possible removed from any condition resembling hysteria.

My own results in operation have been almost all failures, so far as absolute cure is concerned. I do not know that I can point to more than one, or possibly two cases, of what we might call real cure, and even in these there was left generally some slight remnant of the spasm; but in great relief of the condition, a considerable proportion have been successful. Even in this case I allude to, which was one of the worst I ever saw, the man, a stout hearty fellow, recovered sufficiently to resume his occupation after both operations had been done.

The operations have been referred to as heroic or very radical. It is true in one sense that they are heroic or radical; a great deal is done, a number of nerves are cut or excised, a number of muscles are affected. But we ought to remember that these operations are not dangerous. I have never seen the slightest ill result from any operation I have done; and I have never seen a patient placed in danger of life from it. I have removed a large number of nerves, both anterior and posterior; and I have never had any other than good recoveries. With the resources of modern surgery I do not hesitate for a moment in recommending the most radical measures.

It is a radical disease; the patients are willing to have anything done, for life has become a burden to them.

As a rule I begin with a resection of the spinal accessory nerve; and this resection has been a disappointment to me in a recent operation that I did for Dr. Weir Mitchell. The posterior muscles did not seem to be involved; the nerve was resected, and yet the patient was not cured, and I do not know that she is very much better. I think, perhaps, a second operation for the division of the posterior cervical nerves may give relief. If necessary, I should not hesitate to resect the entire sternocleidomastoid as well. The posterior muscles are difficult, mechanically, to resect, and call for the best anatomical and surgical knowledge of the surgeon; but this resection is not impossible for anyone to perform, and no grave results are to be feared.

Dr. V. P. Gibney.—I quite concur with the last speaker. The subject of torticollis has interested me very much for a number of years, but my experience has been largely in the torticollis occurring in young children, or in adolescents, and not especially of the nature under discussion at present. I take it from the remarks that I have heard this morning that it is this rotary, spasmodic torticollis that has proved so obstinate in resisting treatment. I have had a number of cases myself, and I want to say that the torticollis occurring in children and adolescents especially, is that which dates from some injury to the sternocleidomastoid chiefly, and in these cases you rarely, if ever, have any of these grave disturbances. These cases are usually relieved by section of the muscles.

In regard to the more severe cases, I am just as frank as the rest of you to say, I know of no form of apparatus that offers as much relief as has been suggested or has been obtained by the absolute rest treatment or the nerve and muscle section. I have often thought that if after partial division of the muscles or stretching of the nerves, the neck could be retained for two or three months in a rigid plaster of Paris dressing, that this habit might be overcome, and that much relief might be afforded. I have employed this in two instances, but was unable to keep up treatment sufficiently long to make any assertion as to its curative effects. I reason by analogy to spastic disease of the limbs, and the gratifying results obtained from prolonged retention of the limbs in rigid plaster dressing. It seems to me that if these cases of torticollis could be so retained much good could be accomplished. It may seem that it is almost as severe to leave the patient in plaster of Paris as in bed absolutely immovable; but I can add my testimony to Dr. Keen's about operations, and if plaster of Paris is well applied to the head and to the thorax, leaving the arms quite free, the patient can go about very readily. It is not an uncommon thing in the operations at the hospital with

which I am connected to divide both the cervical and clavicular ends of the muscles, and put the child up in plaster of Paris then and there, and on the second day have the child up and about, and in fact in one case we had a child operated on on Wednesday, who went to the circus on Thursday or Friday.

In regard to gelsemium, I myself, a number of years ago, when Dr. Mitchell brought this subject up, treated the case of a boy with rotary spasm of the neck with most gratifying results, having reached toxic doses. I have since tried it repeatedly in adult cases, and never had any results whatever, so that this case of mine is unique so far as my experience goes.

Dr. J. J. Putnam.—The subject of the therapeutics of torticollis has been discussed pretty fully, but there is one part of it to which allusion has scarcely been made, and that seems to me to be the most important, on the whole, and that is the methodical education of the muscles, or co-ordinating centres. It seems to me that even where the surgical treatment is used this education of the muscles is an essential after-treatment. Dr. Walton referred to massage as something that might be tried, but on the whole not amounting to much. Massage, however useful it is if given by a skilful person, is really only one part of this method. I know of one patient who cured himself by an effort of the will and gradual training. My colleague, Dr. Coggeshall, has reported excellent results in the way of absolute cures. The method of Kocher, though ostensibly surgical, is really based on a system of muscular training; the muscles are divided instead of the nerves, and although the treatment acts as a suggestion to the nervous system, still, it is specifically said that the suggestion is only a part of an educational method. The objection to this method is that it takes time, many months, and possibly several years, to get a satisfactory result; but during that time the patient is slowly and satisfactorily improving, if he is willing to devote the proper kind of attention to it.

Dr. Langdon.—My experience with this affection has been exceedingly limited, but I call to mind one case in particular which presents certain points that may be of interest in connection with this subject. First, with regard to the question of cause. The patient was a trolley car conductor, having in the course of his occupation to bend his head back in order to adjust the pole of the car on the wire. He had a very violent form of spasmodic torticollis, according to my diagnosis, and on account of its sudden onset and the youth of the patient—he was twenty-two or twenty-three—I was inclined to take a rather favorable view of it, and in accordance with that view two or three electrical applications were attended with very remarkable curative results for a time; but the spasm would come on worse than ever in the course of a few hours. After

giving him the usual course of sedative treatment, with the exception of gelsemium, and without improvement in the symptoms, I was almost inclined to think that there was a gross lesion connected with the vertebral column. He went to the hospital, where they blistered him and used various other treatments, but with no result. Then I adopted the plan of treatment which I had seen used by Bastian in hysterical contracture with good results; I put him to bed; kept him on milk diet and asleep with bromide and chloral sufficient to stupefy him for three weeks. He went out of the hospital perfectly well, and I think it likely that he remains well since. If a return of the spasm should occur, I think the patient would be very apt to report promptly for further treatment.

Dr. W. M. Leszynsky, of New York.—Some of those present may remember that I published some years ago an account of a case of clonic torticollis that was cured by the subcutaneous injection of atropine in increasing doses. I also reported an analogous case of clonic spasm affecting the platysma which was cured in the same manner.

A lady thirty-five years of age has been under my constant observation during the past eighteen months. The clonic spasm had previously existed for six months and involved the sternomastoid, trapezius and splenius capitis. The sternomastoid was hypertrophied to nearly four times its natural size. Atropine treatment could not be continued owing to idiosyncrasy. Conium and gelsemium failed. After prolonged rest and tonic treatment, together with local massage, passive movements and educational gymnastics faithfully and persistently carried out, the clonic spasm has been absolutely cured, having been absent for nearly ten months. This has, however, been replaced by a mild form of tonic spasm remittent in character and involving the trapezius and splenius. The sternomastoid has resumed its normal condition. A peculiar feature was the high specific gravity of the urine, which was due to urates. Whenever it rose to 1030 or 1034 the spasm increased, seemingly indicating an autotoxic irritative element in this case. It seems to me that the most important point is to treat these patients early, by prolonged rest in bed, massage, etc., and if this were done fewer cases would advance to such a stage as to require surgical operation.

Dr. Prince.—Regarding the efficacy of the surgical treatment, it seems to me that enough patients have been treated in this way to prove that it is efficacious. It certainly seems to me a rational treatment, and if there is no other way of curing them, I for one should not hesitate to advise the radical surgical operation. But while giving full credit to this means, I do think that Dr. Walton has not given equal credit to other methods of treatment, which should be tried first.

I have never seen any beneficial results follow the drug treatment. I have tried in three or four cases the atropine treatment, pushed to intoxicating doses, but I was disappointed in each case in obtaining no result. On the other hand, very beneficial results were obtained in one case, of which I have knowledge, by the use of massage, intelligently applied, as referred to by Dr. Putnam. The case had been treated medically by me without benefit, and I felt justified in referring it to a surgeon for operation. Fortunately he took a conservative view and preferred to try the massage treatment first, and she was put in Dr. Coggeshall's hands for this purpose. Dr. Coggeshall not long since told me that the treatment had been successful, and that she is now perfectly well.

On the other hand, there are certain cases of a moderate severity which, although not curable by drugs, or rest, or anything else, yet are not sufficiently severe to warrant surgical operation, and get along with some sort of apparatus.

Regarding the nature of this affection, it seems to me that all analogy shows that these cases are, as has been suggested, pathologically very similar to, if not identical with, the occupation neuroses. A case of my own is suggestive of this fact; namely, a young woman who not only had severe torticollis, but also had occupation neurosis, piano-player's finger. Taking writer's cramp as a type of the occupation neuroses, analysis shows that one form, at least, consists in the overaction of certain muscles which have been combined with one another as a result of education. The spasm must represent the overactivity of a physiological process, so that it may be looked upon as quasi-physiological. Over-education, so to speak, has produced it, instead of the muscles contracting with a delicately balanced force, they do so with an excessive force. From the point of view from which I am speaking, it does not matter whether the reason for this be in the muscles or in the centres. I am inclined to believe it is in the centres, but however this may be, the spasm is an excessive contraction forming part of an associated group of movements, and therefore quasi-physiological. Now, it is highly probable that the only difference between torticollis and the spasm of writer's cramp is that in the latter the exciting cause (attempts to write) are intermittent, and therefore the spasms are intermittent; while in torticollis the exciting cause is continuous and the spasms are likewise continuous.

Returning to treatment, it would seem, theoretically, that by rest and the education of a new set of movements, we might accomplish something. This, of course, is easier to do in writer's cramp than in torticollis, for obvious reasons. I can recall one case of writer's cramp which I cured by giving the patient an apparatus which held the pen by the use of the an-

tagonistic movements; that is, by extending the fingers instead of flexing them. The patient can now write as well as ever. It seems to me that the combined treatment of torticollis by education, massage and rest should be tried before resorting to an operation.

Dr. Wharton Sinkler.—To my mind, there are three distinct types of spasmodic torticollis; first, the hysterical type; secondly, the type dependent upon affections of the spinal accessory nerve outside of the foramen; and thirdly, that which is cortical in its origin. The hysterical form very closely simulates the organic form. The intermittency of movements, the position of the head, and the muscles involved, all make it in outward appearance identical with organic forms. This type is very readily cured by proper treatment.

There is also a variety of spasmodic torticollis which is the result of irritation of the spinal accessory nerve outside of the foramen, and which results from over-use of the neck muscles, and I think undoubtedly at times this is a form of occupation neurosis. The treatment which is most successful is absolute rest in bed, with properly applied massage, and educational movements of the muscles. There are two remedies which I have found to give distinct relief; these are gelsemium, pushed to its physiological limit, and conium. I have treated at least two cases by conium in which complete recovery resulted.

In cases in which the ordinary medicinal means, rest, and proper exercise of the muscle fail to relieve the affection, I think we must be driven to the conclusion that there is a cortical lesion, and in such cases I do not think that, as a rule, any surgical interference is of much value. I have seen a number of patients who have been subjected to excision of the spinal accessory nerve, and in some the cervical nerves have also been divided, but with unsatisfactory results in nearly every case. The patient to whom Dr. Keen referred was, I think, a patient of mine, a blacksmith, who was a large and vigorous man; both spinal accessories were divided with absolutely no benefit, and later, the cervical spinal nerves were cut, but the movements of the head persisted.

Dr. W. W. Keen.—I think in his last statement Dr. Sinkler is in error. The patient declined any cortical operation because he was so much better. To be sure there was no immediate benefit, but the man was able to return to his work. The head was not still, but it was relatively quiet.

(To be continued in the November number.)

A CASE OF DYSPHAGIA AND DYSPHASIA RESULTING FROM A LESION IN THE INTERNAL CAPSULE.¹

By JUDSON DALAND, M.D. (UNIV. OF PENNA.), PHILADELPHIA;

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This patient, a married man, fifty-six years of age, was seen in consultation with Dr. W. H. Warder on September 25th, 1896, when the following history was obtained: There is nothing of importance in his family or personal history until five years ago when he suffered from repeated attacks of gout or rheumatism, which more particularly affected the left foot. Soon afterwards he was considerably shocked by the death of his father, and later was much worried by business troubles. Upon April 8th, 1894, at one A. M., he awoke with intense pain in the back of the head, and shortly afterwards, while evacuating the bowels, he fainted. This intense pain continued several weeks despite treatment. Seven months later he had a similar, though milder attack. Two years and four months ago he was under the care of Dr. Joseph Leidy, Jr., and at this time, while evacuating the bowels, his wife found him mumbling in an unintelligible manner, which made it impossible for her to understand what he desired to express. He was able to walk, and there was no paralysis of the arms or legs. There was interference with the movement of the tongue and the left side of the face with considerable dysphagia, which seemed to be dependent upon the condition of the pharyngeal muscles, especially the constrictors. There was

¹Read before the Philadelphia Neurological Society, April 26th, 1897.

no true aphasia. He made a complete recovery in four weeks. About this time he was subject to attacks of poly-articular rheumatism, and from time to time suffered with indigestion. About fourteen months ago he developed dyspnea with marked irregularity of respiratory movements, which later became pronounced Cheyne-Stokes breathing. This was accompanied by edema, more especially of the lower extremities, and at that time his physician recognized the existence of cardiovascular and renal disease.

At the time of my first examination Cheyne-Stokes breathing was well marked. There was moderate edema of the lower extremities. The heart showed considerable hypertrophy of the left ventricle, but the sounds were clear and all arteries accessible to touch showed well-marked thickening from fibrosis and atheroma.

A summary of twelve urinary analyses shows that the quantity varied between thirty-three and forty-three ounces, that the color was lighter than normal, that the albumin varied from one to two per cent., that there was no sugar, that there was usually present slight indicanuria, that the specific gravity varied between 1008 and 1011, that the reaction was decidedly acid, that the urea was 8/10ths of one per cent., and that at first no, and later a few, narrow hyaline tube casts were present. Shortly before death the hyaline tube casts increased in number, and a few granular casts were also found.

The lungs showed a moderate grade of emphysema. The digestion was feeble, and he was limited to the simplest food. Any slight indiscretion in diet would produce marked gastric disturbance and even nausea. The liver was slightly enlarged. The speech was at times hurried and enunciation somewhat mumbling, but a part of this defect in speech was due to rapid and irregular breathing.

The eye-grounds were repeatedly and carefully examined by Dr. Thomas H. Fenton, who found that the nerve heads were slightly elevated and pale, that the veins were dilated, and that the arteries were contracted. There

were no disturbances of the visual fields, and acuity of vision was normal.

Cheyne-Stokes breathing continued more or less well marked until five or six weeks before death, when it suddenly disappeared. He died suddenly on January 26th, 1897, from heart failure.

The autopsy was performed Jan. 28th, 1897, with the following results: The heart and blood vessels attached weighed nineteen ounces, and the heart was very much enlarged in all its diameters. The left ventricle, which was



FIG. I.

I, Caudate Nucleus; *J*, Small Lesion in the Caudate Nucleus; *K*, Thalamus.

dilated and hypertrophied, constituted the major part of the cardiac enlargement. The mitral valves and orifice were normal. All visible arteries showed marked atheromatous changes. The inferior vena cava was of large size and partially empty. The pericardium was normal.

The aortic leaflets were competent, moderately thickened, but otherwise normal. The tricuspid orifice was dilated, but the valves were normal as were also the pulmonary valves. At the extreme lowermost portion of the left ventricular cavity, at a point corresponding to the apex, were a moderate dilatation and thinning of the walls, and the ventricle contained an organized thrombus of about

the size of a large marble. Superimposed was a small and more recent thrombus. Some of the chorda tendineæ attached to the mitral valve were attenuated and elongated. The base of the anterior leaflet of the mitral valve, near the base of the aortic leaflet, showed several patches of atheroma which were yellow in color. The aorta was thickened and dilated, and pouched posteriorly and to the right. There were numerous atheromatous patches beneath the intima. The spleen was at least three times its normal size, and was adherent posteriorly. The structure showed no macroscopical change. The left kidney

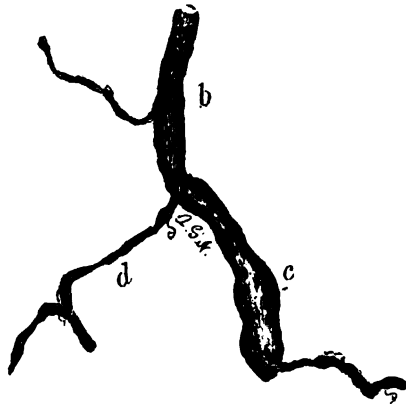


FIG. II.

b, Basilar Artery; *c*, Right Vertebral Artery; *d*, Left Vertebral Artery.

was smaller than normal, and the capsule was adherent, but with care could be detached from the cortex without tearing the renal structure. The entire surface presented a markedly granular appearance, and the superior portion was occupied by a large cyst the size of a walnut. Over the surface were two or three smaller cysts containing a clear liquid. Upon section the cortex was narrowed and showed the presence of a large amount of fibroid tissue. The pyramids were smaller than normal, and showed marked fibroid changes. The ureters were normal. The

right kidney was in a similar condition. The condition of the kidneys was the result of a long-continued, chronic, interstitial and parenchymatous nephritis. The gastrointestinal tract showed no abnormality.

The liver was enlarged and projected four fingers' breadth below the edge of the ribs. On section it presented the characteristic nutmeg appearance, and showed evidence of considerable fibrosis and moderate fatty degeneration.

The calvarium was unusually thick anteriorly. The sinuses were empty, and there was no evidence of inflammatory change in the meninges. There was a moderate amount of effusion beneath the meninges and within the ventricles and the spinal canal. The convolutions of the brain were well developed and appeared normal. Upon section the white substance appeared normal, and the red spots corresponding to sections of dilated vessels were numerous and of considerable size.

About the middle of the caudate nucleus and cutting or pressing upon the fibres passing to the knee of the right internal capsule was a lesion of about $\frac{1}{4}$ of an inch in diameter and $\frac{1}{8}$ of an inch in depth, which showed the remains of a hemorrhage which probably occurred two years and four months previously. This lesion was plainly apparent on opening the right ventricles, and is well shown in Fig. I.

The blood vessels all presented marked evidence of atheroma. The right vertebral and basilar arteries, especially the former, were aneurysmally dilated and pouched, and their calibre was fully as large as an ordinary carotid (Fig. II.). In places the wall was extremely thin and almost transparent. This aneurysm was close to the medulla oblongata and was quite capable of causing a certain degree of pressure upon the neighboring tissues. The left vertebral was unusually small. The posterior meningeal arteries showed atheroma, but no marked change in the lumen.

This case is interesting as showing (1) that a small

superficial lesion situated close to the knee of the right internal capsule is capable of producing dysphagia and dysphasia; (2) the appearances presented by a small hemorrhage two years and four months after its occurrence; the symptoms produced and their variations during this period; (3) that the wide-spread and advancing arterio-capillary fibrosis and atheroma, probably rheumatic in origin, produced the customary cardiovascular and renal changes, and also the aneurysm of the right vertebral and basilar arteries; (4) the long continuance of the Cheyne-Stokes breathing on and off for four or five months, which was probably in part due to cardiac failure and slight uremia.

The aneurysm of the vertebral and basilar arteries, pressing upon the medulla, raises the question as to whether this also may not have been a contributory factor in the production of the symptoms.

The situation of the small aneurysm was such that it could readily have compressed the right glossopharyngeal and hypoglossal nerves, so that the difficulty in swallowing could in part be explained in this manner.

PARALYSIS OF RIGHT THIRD NERVE FROM HEMORRHAGIC NEURITIS
WITH EXTRAVASATION OF BLOOD OVER THE OPPOSITE FRONTAL
LOBE.

In the *Edinburgh Medical Journal*, for May, 1897, Gibson and Turner report the case of a girl eleven months old, admitted to the hospital, for diarrhoea and vomiting, with wasting, who had right-sided ptosis, with dilation and immobility of the pupil, and inability to turn the right eye inward. Her temperature ran an irregular course, her pulse and respiration were rapid, and she died after five days, having presented no more definite symptoms. The autopsy showed a hemorrhagic neuritis of the right third nerve, the left being nearly normal. On the left side of the brain there was a large hemorrhage, occupying the area over the middle third of the Sylvian fissure, with a prolongation upwards and forwards involving the bases of the frontal gyri, especially that of the second. The centrum ovale and gray matter about the Sylvian aqueduct showed nothing abnormal. The authors regard the nerve lesion as entirely responsible for the eye palsy. No mention is made of the condition of other organs. C. L. ALLEN.

HEADACHE WITH VISUAL HALLUCINATION.¹

By JOHN K. MITCHELL, M.D.

The description of the following very extraordinary case properly begins with a history which presents some points of interest. The patient, Mr. S. G., is a native of the Spanish West Indies, and is one of nine children, of whom seven survive, two having died in infancy. Both parents are living; his father is gouty, but there is no history of neurotic disorder in the family, except that one uncle is said to have been impotent and to have committed suicide. The patient is himself married and has two children. He can recall no attack of illness, except measles in childhood. He has led a temperate life, and his habits have been good, with the exception of an excessive use of tobacco in the form of cigarettes. He is a lawyer and in active and successful practice.

Mr. G.'s appearance is bad; he is sallow, looks anemic, has a melancholy and depressed expression. He generally sleeps well, and his functions are said to be normal; the appetite is good; the heart and lungs present no abnormality. The urine has been repeatedly examined without result, both here and at his home. His complaint is of increasing recurrent headache, now of 'three years' duration, accompanied by an apparition of a very curious sort, and followed by blindness and finally by loss of consciousness and violent convulsions. He is usually aware of the approaching attack by beginning to lose his sight. When this symptom appears he goes to bed, and the attack pursues a regular course. There is first a vision of a tiny

¹Read before the Philadelphia Neurological Society, April 26th, 1897.

dwarf, half an inch high, appearing at a great distance; he gradually approaches, becoming larger and larger, until he assumes the form of a gigantic gladiator with bared limbs, and armed with a club. During his approach the pain in the head is constantly growing worse, beginning with fleeting painful sensations of variable location, sometimes parietal, sometimes vertical, never occipital, frontal or supraorbital. When the giant comes close, he strikes the patient repeatedly on the head with the club, producing excruciating and terrible pain, which increases with each stroke until there is loss of consciousness, and then violent convulsions usually follow. In the convulsions he is drawn backward, arching the body upward, and even resting on the head and heels. The time from the first appearance of the dwarf until he strikes the patient's head has been as much as eight hours, but it is usually somewhat less. The duration of the attack was at first about twenty-four hours, but with returning and more frequent attacks, the time which they last has decreased to about eight hours; the worst pain and convulsive seizures last from fifteen to forty minutes. Afterward the teeth feel on edge, he is somewhat sore all over, but otherwise pretty well.

Of course, the eyes were supposed at fault, and examination was made by his own physician, and again by Dr. Chas. A. Oliver, during Mr. G.'s stay here. Dr. Oliver's report is as follows:—

“Examination, Dec. 7th, 1896, shows the vision of the right eye reduced to one-tenth of normal, though brought to full acuity by the use of a very high concave cylinder lens. The vision of the left eye is normal.

“Power of accommodation greatly disturbed in the uncorrected right eye; brought to normal by the use of the cylindrical lens.

“Fields of vision for colors and form properly shaped and sized. Muscle balance undisturbed in the two eyes, either in the vertical or horizontal meridians or during near or distant vision.

"Pupils the same size and irides freely and equally mobile to light, convergence and accommodation.

"The ophthalmoscope shows an absolutely healthy fundus in both eyes.

"The error of refraction and accommodation in the right eye has been fully corrected, restoring the two eyes to the same degree of physiological power and action."

From this it may be concluded that, although a wholly unilateral refraction-error of so extensive a degree may be unusual, it is not very likely to have been the exclusive cause of such a form of cephalalgic pain as is here presented.

Apart from the visual hallucination, the most peculiar feature of the case is that the attacks never occur during the winter, but only from May to December. They have completely ceased since he arrived in this country in November. At first there was five months' interval between the seizures, then two or three, then about fifteen days, and of late they have occurred as often as every five days. Mr. G. says that on one occasion, and only on one, he was able to converse with the apparition, and ask him why he tortured him so? to which the giant replied, "he had been ordered to do so, and would continue while Mr. G. remained in the country."

So far as I am able to discover, the first reports of cases of the apparition of a human figure, in connection with headaches, were among those related by Dr. Weir Mitchell in the *Transactions of the College of Physicians* for 1887. Two years later, Dr. De Schweinitz added six cases of visual hallucination. Dr. Mitchell's patients all suffered with headache of the hemi-cranial type; Dr. De Schweinitz's were of various sorts, some of them migrainous, all of them marked by severe pain. None of those related by either gentleman exhibited the peculiarity of this case in having a causal relation between the spectral illusion and the pain. Some of them had visions of animals or human beings; some the more

common appearance of zig-zag-colored lines, or fireworks.

The hallucinations of migraine occur sometimes as precursors of the pain; sometimes during the height of the attack. The only instance within my knowledge of an hallucination other than visual as an accompaniment to cephalalgia occurred in my own practice, and is briefly as follows:—

A lady, twenty-five years of age, in good health, and with no neuropathic taint in her family history, but inheriting strongly marked gouty tendencies, suffered at intervals of a few weeks with violent neuralgic headaches, the pain being felt in the frontal and orbital regions, and only rarely culminating in nausea and vomiting. Treatment was almost useless, and the attacks usually persisted for some hours, little affected by any drug, even hypodermic injections of morphia giving but slight relief. So severe was the pain that she was not infrequently quite delirious. At the height of the attack she would suddenly perceive a very strong and distinct smell of violets, a sign which was always received with joy, because almost from that moment the suffering would begin to diminish. A lingering sense of the odor of violets was present with her sometimes for more than half an hour, by which time relief was generally very great. The attacks became less frequent and much less violent after a long and severe siege of typhoid fever, and have never since been so bad as before; nor, I believe, has the smell of violets ever been noticed since the fever.

It is interesting to note in connection with the violence of the headaches from which this patient had suffered for some years that the onset of typhoid fever was marked by headaches, frontal and occipital, with constant retraction of the head, and of such severe character as to almost completely mask the symptoms of the fever itself, and to cause doubt in the minds of the consulting physicians, Dr. Da Costa and Dr. Weir Mitchell, as to the diagnosis, and make them seriously consider the possibility of menin-

gitis. Dr. J. C. Wilson, the attending physician, always held the belief that the case was typhoid, and this opinion was later justified by the course of the disease.

The recurrent form in which Mr. G.'s headaches appear naturally suggest the possibility of malaria. The patient has never suffered with malarial infection in any form; nor does he present any symptoms which could be thought due to that disease. No protozoa have been found in the blood; the spleen is not enlarged, and malarial fevers are not very prevalent in his place of residence.

The diagnosis of hystero-epilepsy has been made in this case by a physician at his home—a suggestion to which a certain color is lent by the convulsions which accompany the seizures, which are described as typically opisthotonic. It has been held too that the attacks were truly epileptic. But as he does not bite his tongue, and as he preserves his consciousness until after the pain has lasted for some little time, and as he comes at once and without any subsequent heaviness or drowsiness out of the attacks, this does not seem possible. The gradual approach of the dwarf, and the way in which his arrival determines headache, resemble slightly a patient described by Dr. Weir Mitchell, whose visual delusion was that of the distant appearance of a point of light gradually approaching, and when it reached the patient, exploding, at the instant when the attack of pain began.

My belief about this patient is that the headache is a peculiar expression of that extraordinarily various disease, migraine. I found this opinion first upon the order of occurrence of the phenomena. In migraine it is usual to see first slight visual, or other sensory disturbance; second, pain; third, the special form of nerve-storm which the headache takes in the individual patient. In Mr. G.'s case the sequence is, first, slight disturbance in the form of visual hallucinations; second, pain, which goes on increasing in violence until it ends in a nerve-storm, namely, the convulsive seizure described.

Second, periodicity of attack is another feature often present in migraine; malaria, as has been stated, is excluded.

Third, the progressively culminating character of the disorder belongs likewise to the migraine type.

Fourth, epileptiform convulsions are not unknown in migraine, and a distinct migraine is even sometimes replaced by pure epileptic fits.

The refractive error, although perfectly corrected, very probably has its share in the production of trouble. It may not be too fanciful to think that an extra sensitiveness to light (due to refractive error) may add its quota to the strain upon the nervous system, especially when it is remembered that the attacks occur only during the months of greatest warmth and sunshine.

The attacks determine in some fashion which we cannot understand, changes, probably circulatory, in the cerebral centres presiding over vision, which result in the spectral apparitions.

It is as yet impossible to say what effect treatment may have. No report has been received from the patient since his return home, and he was last seen during the months of freedom.

CHANGES IN THE CORD FROM STAPHYLOCOCCUS INFECTION. *Gazette Hebdomadaire de Médecine et de Chirurgie*, April 7th, 1897. *Société de Biologie*, March 13th, 1897.

Roger and Josue observed a case of infection by staphylococcus, and were able to verify in man the results which they obtained in studying the cord of animals experimentally inoculated.

The case was that of a young woman, 19 years of age, who died in 15 days of multiple abscesses of the skin of the head and of the nape, with foci of broncho-pneumonia and double purulent pleurisy; the entrance of infection having been an abundant pityriasis. All the foci were due to staphylococci.

Sections of the marrow examined by the naked eye appeared red; while in the normal state the tissue was so delicate and transparent that one could see it with difficulty.

The changes were analogous to those observed in a rabbit 48 hours after inoculation of staphylococci under the skin. The cells, which had multiplied, were of the same variety, but the process was slower, as it took 15 days to develop the condition in man.

MITCHELL.

THE NEUROMUSCULAR BUNDLES (MUSKELKNOSPEN, MUSKELSPINDELN, FAISCEAUX NEURO-MUSCULAIRES).¹

By WILLIAM G. SPILLER, M.D..

Associate in the William Pepper Clinical Laboratory; Professor of Diseases of the Nervous System, in the Philadelphia Polyclinic.

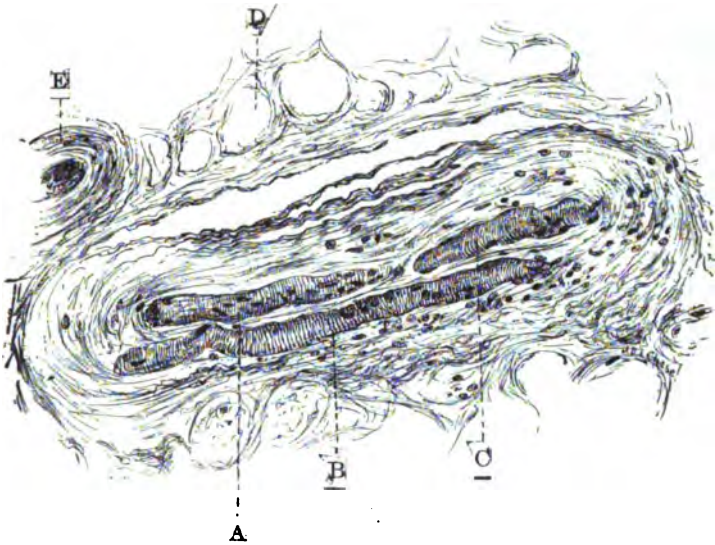
From the Salpêtrière, Paris (Laboratory of Professor Dejerine), and the William Pepper Clinical Laboratory.

The excellent state of preservation of the neuromuscular bundles in a case of intense muscular dystrophy studied microscopically by the author seems of sufficient importance to warrant a brief statement, especially as very little has appeared in the English literature on this subject.

The case was one of intense muscular atrophy with contractures; it was from the clinic of Prof. Dejerine at the Bicêtre, Paris, and has been referred to by him in his lectures. No lesions were found in the spinal cord which could in any way explain the amyotrophy, for the ganglion cells were well formed and were about normal in number. The deltoid muscle, of one side at least, was so intensely atrophied that it appeared more like a portion of the diaphragm, and when examined microscopically, was found to be almost entirely composed of connective tissue. In a few sections from this muscle, in one portion of the field, a single group of hypertrophied muscular fibres could be seen, which had undergone hyaline degen-

¹This case was reported and the specimens were exhibited at a meeting of the Philadelphia Neurological Society in 1896. The paper was already in type when the work of Batten on the neuromuscular bundles (Brain, Parts LXXVII. and LXXVIII., 1897) came into the hands of the writer.

eration and longitudinal segmentation, as described by Erb. Elsewhere it was impossible to find anything which in any way resembled muscular tissue, except in the neuromuscular bundles. The intramuscular vessels were much thickened, but the small intramuscular nerves were apparently perfectly normal. The medullary sheaths of these nerves, when stained by the method of Weigert, had the appearance of normal fibres, and when the carmin was



Longitudinal section of a neuromuscular bundle.

A B C, small muscular fibres within the bundle. D, fatty connective tissue which has replaced the surrounding muscular tissue. E, small intramuscular vessels with greatly thickened coats.

employed the axis cylinders were distinctly seen. In some microscopic fields two neuromuscular bundles could be seen, whereas in others not a single one could be found. It has been possible to obtain several good longitudinal sections of these structures, and in one section a bundle was cut in such a manner that it afforded a transverse and longitudinal view of the same muscular fibres; a result which was due to a curving of the bundle. The external

sheath was very thick in comparison with the intramuscular nerve sheaths, and within this were found connective tissue rich in nuclei, and one or two delicate muscles cut longitudinally. The nuclei of the muscular fibres were slightly elongated and quite numerous, but they did not appear to be more abundant in the centre of the fibres. In the picture of a longitudinal section, published with this paper, it may be seen that these muscles are of considerable length for such delicate structures; that they are of less diameter than are ordinary muscular fibres, and that they are transversely striated. The state of preservation of the fibres as compared with that of the surrounding muscle is most striking.

Excellent pictures of *transverse* sections of neuromuscular bundles have been published, and two especially good are to be found in an article on neuritis, by Babinski. ⁽²⁾ Laura Forster ⁽³⁾ has carefully studied these bodies, and is able to refer to quite an extensive literature on the subject. According to her statements, Weismann ⁽⁴⁾ was the first to note their existence. Koelliker ⁽⁵⁾ described them in 1862 as "*Nervenknospen*," a name which he has now changed to "*Muskelknospen*."

Among other investigators may be mentioned Aeby, Kühne, Ranvier, Krause, Bremer, Mays, Kerschner, Blocq, Marinesco, and Sherrington.

Babinski ⁽⁶⁾ believed he was the first to describe the neuromuscular bundles as found in man, although Roth had already reported to the Medical Society of Moscow the results of his investigations in this field. Kerschner and Felix showed that the neuromuscular bundles of Roth

²Babinski: *Traité de Médecine*, VI., p. 704.

³Laura Forster: *Virchow's Archiv*, 1894; 137. p. 121.

⁴Weismann: *Zeitschrift für rationelle Medizin*, 1861, Vol. X., p. 263.

⁵Koelliker: *Gewebelehre*, Vol. I., p. 394.

⁶Babinski: *Comptes rendus hebdomadaires des séances de la Société de Biologie*, 1886, p. 629; and *Archives de Médecine Expérimentale*, 1889, p. 416.

and Babinski, found in man, are the same as the muscle spindles of the German writers, found in animals.

Weiss and Dutil (⁷) describe the nerve fibre of the neuromuscular bundle as terminating by free ramifications, some of which embrace the large nuclei of these small muscles. At the enlargement of each muscular fibre is a group of nuclei two or three times the size of the nuclei of ordinary muscles, and at this part the muscular striation usually ceases; in other parts of the fibre scattered nuclei are found.

Weiss and Dutil employed the chlorid of gold in their investigations. They found the neuromuscular bundles especially numerous near the tendons, and they state that in some cases it may only be possible to distinguish them from the corpuscles of Golgi by the transverse striations. The same nerve differing, as regards size, number of nodes, and thickness of the sheath of Henle, from the ordinary nerve of the surrounding muscle, sends branches to the neuromuscular bundle and the organ of Golgi. The muscular bundles contain motor plates, similar to the motor terminations in the ordinary muscle, and this fact they think proves the power of contractility of these small fibres.

Comparatively few histologists of the present day hold the opinion, once quite prevalent, that these bundles are pathologic, for, as Sihler (⁸) justly remarks, their construction is so evidently adapted to some special function that any such view is improbable. Some have held that they are ordinary muscular fibres in the process of formation, although no one has shown the intermediate stages; others that they indicate retrograde change. Kerschner (⁹), Pilliet (¹⁰), Laura Forster, and others, have ascribed

⁷Weiss and Dutil: *Archives de Physiologie*, April, 1896.

⁸Sihler: *Archiv für mikroskopische Anatomie*, 1895, 45, p. 709.

⁹Kerschner: *Anatomischer Anzeiger*, 1888, 1893.

¹⁰Pilliet: *Comptes rendus hebdomadaires des séances de la Société de Biologie*, 1890, p. 313.

to them a sensory function, and Weiss and Dutil have expressed themselves in favor of this view. This function seems to stand in some special relation to the muscular sense. Golgi has thought that they are connected with the lymphatic system.

• Roth (¹¹) has found these bodies in progressive muscular atrophy and other diseases; Pilliet has studied them in alcoholic paraplegia, chronic rheumatism and amyotrophic lateral sclerosis; Blocq and Marinesco (¹²) in poliomyelitis and polyneuritis; Eichhorst (¹³) in neuritis; Laura Forster in myelitis; in a case of progressive muscular atrophy in which no definite lesions were found in the cord, and in bulbar paralysis. Professor Dejerine has kindly called the writer's attention to the fact that Roth, in his large work on muscular atrophy, published in Russian, speaks of them again in primary atrophic myopathy.

The more intense the atrophy, the more distinct these bundles become, and in the case of atrophy from myelitis, reported by Laura Forster, in which many nerves were degenerated and the muscles were almost entirely destroyed, these bundles were well preserved.

Whatever may be the opinion in regard to the nature of these bundles, it is well worthy of note that in complete degeneration of muscular dystrophy, when the intramuscular nerve fibres appear normal, these neuromuscular bundles may undergo no notable change.

The pleasant duty of thanking Professor Dejerine for the pathologic material and for many manifestations of kindness is gladly discharged by the writer.

¹¹Roth: *Centralblatt für die medicinischen Wissenschaften*, 1887, p. 129.

¹²Blocq and Marinesco: *Comptes rendus hebdomadaires des séances de la Société de Biologie*, 1890, p. 398.

¹³Eichhorst: *Virchow's Archiv*, 1888, CXII.

A CASE OF PROGRESSIVE NEUROTIC MUSCULAR ATROPHY.

By CHARLES W. BURR, M.D.

History.—L. A., male, 31 years old, a clerk, applied for treatment at the Medico-Chirurgical Hospital in October, 1896, complaining of a slowly increasing difficulty in walking and weakness in the hands. His family history is negative. His previous health has always been good. He has not had gonorrhea or syphilis, and has never used alcohol. His sexual habits are good.

The present trouble began when he was in his twenty-sixth year, with severe pain in the left heel soon extending to the left calf. In three or four weeks his left ankle began to be weak, and pain came on in the left knee and hip. A year later he noticed weakness of the right lower limb without preceding pain. A few months after the onset he noted wasting in the left calf, and later (after the weakness) in the right. Wasting and weakness of the hands began several years after the legs were affected, and was not preceded or accompanied by pain or paresthesia. He has never had any loss of control of the bladder or rectum. His sexual power is good.

Examination.—The gait is waddling. He cannot stand with the feet close together, because of weakness. He cannot extend the toes, and can extend the feet only weakly. There is neither club foot nor other deformity. There is marked weakness in all the movements of the hands. Extension of the forearms is good, but flexion is rather weak. The shoulder and hip movements are normal. There is great wasting of both calves, some of the thighs, especially the left, and a little of the intrinsic muscles of the

feet. The muscles of the hands are much wasted, the forearms somewhat, the upper arms little, if any; and the shoulders, neck and face not at all. The knee, biceps tendon, cremasteric, and muscle jerks are absent. There are no fibrillary twitchings. Sensation of touch, pain and temperature is normal. There are no vasomotor symptoms.

The first description of progressive, neurotic atrophy was given by Charcot and Marie in a paper in the *Revue de Médecine* for February, 1886. A short time later Howard Tooth published a thesis describing the same condition, entitled: "The Peroneal Type of Progressive Muscular Atrophy." He quoted cases which had been previously reported by Friedreich, Eichhorst and others, without their having recognised the nature of the affection. According to Charcot and Marie, the disease is characterized by muscular atrophy attacking first the feet and calves, and after considerable time, even several years, affecting the hands and the forearms, but never the trunk nor face. Fibrillary contractions may be present. There are sometimes vasomotor symptoms. Sensation is most often intact, but may be altered in several ways. Reaction of degeneration is frequent. The disease usually begins in early life, often affects several brothers and sisters, and may appear in succeeding generations. Clinical cases have been reported in Europe by Hoffmann, Bernhardt, Vizioli, Donkin, Dejerine and others. The descriptions agree in the main, though differing in detail. For example, Hoffmann has shown that the wasting may appear in the hands first, the legs being affected later. Sensory symptoms may be slight or severe. Thus, Sachs, of New York, says: "Sensory changes are generally present. The various forms of sensation may be slightly altered, or, in some cases, tactile sensation and temperature sense may remain normal, while the pain sense may be more distinctly involved. Paresis may be present in addition to the objective changes in sensation." Gowers states that cutaneous sensibility,

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though often normal, is sometimes impaired or lost, especially over the region in which the atrophy is greatest, or upon the soles of the feet. Pains occur in some cases.



The electrical reactions of the muscles vary, depending in large degree upon the stage during which the examination is made. There may be complete absence of electrical

response, or partial or complete reaction of degeneration, or a mere quantitative diminution.

The only cases reported in America are two described by Sachs, one shown at the November meeting of this Society by Potts, and one not yet reported, to be presented here to-night by Dercum and Leopold, in all, counting this man, five. The Farr family, described by Osler, may, or may not, belong to this group. They were reported before this type of atrophy was distinguished from the others. The affection described by G. Dejerine and Sottas in 1893 (*Mém. de la Soc. de Biologie*), and G. Dejerine in 1896 (*Revue de Méd.*), under the title, "Interstitial, Hypertrophic and Progressive Neuritis of Childhood," is a distinct disease.

Scarcely the first word has been said about the morbid anatomy of progressive neurotic atrophy, nor can we say with any certainty that it is due to the same lesion in all cases. Clinically, it is an entity; pathologically, it probably is not. Muscular atrophy must, so far as present knowledge goes, be due to diseases of the spinal cord, the nerves, or the muscles, since we may disregard the rare cases of atrophy accompanying cortical disease, as being as yet unexplained. Often we can differentiate the three kinds of atrophy. But types shade into each other, and it is often impossible to tell whether a given case be due to nerve disease, muscular disease, or even cord disease. But few necropsies have been made in cases of progressive neurotic atrophy. In the early cases of Virchow and Friedreich, published before the clinical recognition of a distinct neural type of atrophy, there was found degeneration of the nerves and of the posterior columns of the spinal cord, especially affecting the columns of Goll. In 1890 Dubreuilh (*Revue de Médecine*, Vol. X.) reported a case of a child whose mother and eleven brothers and sisters suffered from the same disease. The patient became ill during the second year with a gradually increasing atrophic palsy,

which began in the hands and later spread to the legs. There were no sensory disturbances. Death was caused by tuberculosis. Post-mortem there was found degeneration of the nerves, evidently of long standing, affecting especially the motor nerves of the hands and feet, and diminishing in intensity as the cord was approached. There was a very slight increase of the neuroglia in the columns of Goll and in the lower part of the columns of Burdach. The gray substance was normal. The changes in the muscles consisted of simple atrophy of the fibres, with, in some, a loss of the transverse striations and a proliferation of the nuclei. Some of the fibres were degenerated, a few hypertrophied. This, therefore, was a genuine case. But in 1896 Oppenheim and Cassirer (*Deutsche Zeitschrift für Nervenheilkunde*) reported a case which shows well the great liability to error in the pathological diagnosis of this affection. The patient was a man forty-two years old, and with good inheritance. Within two years an atrophic palsy, accompanied by pain, had developed. The distal parts of the extremities were affected---in the legs the muscles supplied by the peroneal nerves especially and in the arms the small muscles of the hands, and later the triceps and long supinator muscles. There was incomplete reaction of degeneration, and in some muscles no electrical reaction. Sensation was normal. The orbicularis palpebrarum was affected, but no other face muscle. The disease developed slowly, but progressively. Death resulted from angina. During life the case was diagnosticated as either a chronic multiple neuritis or neurotic muscular atrophy. The necropsy revealed only muscular disease. Microscopic examination of the intramuscular nerves, the nerve trunks, and the central nervous system showed practically no pathological condition. The muscles on the other hand showed the changes found in primary myopathies, namely, atrophy of the muscle fibres with occasional hypertrophy, absence of the transverse striation, with the ap-

pearance of longitudinal striation, partial vacuolation, great increase in the connective tissue and lipomatosis. These are not pathognomonic of myopathy, but are characteristic. Hoffmann asserts that there is a primary ascending degeneration of the nerve fibres, which secondarily involves the spinal cord, especially the columns of Goll and the motor cells of the anterior horns.

All things considered, we are probably justified in concluding that disease either of the muscles themselves, or of the nerves, may produce the clinical picture of progressive neurotic muscular atrophy, and that only in individual cases can we, by the accompanying symptoms, the electrical reaction, the presence or absence of sensory and vasomotor symptoms and the like make a correct pathologic diagnosis.

PARALYSIS OF THE OCULAR MUSCLES DUE TO LEAD POISONING.

D. Casey Wood is reported (*Journal of the American Medical Association; Columbus Medical Journal*) to have presented the following case to the Chicago Ophthalmological Society (Feb. 9th, 1897):

Patient, aged twenty-nine, a painter, had lead colic five years ago, followed in two years by an attack of vertigo coming on suddenly one morning, and was then unconscious for half an hour, and had pain through his head for two days, at the end of which time he complained of diplopia and in three weeks noticed that his left eye turned out. There is no history of traumatism or syphilis. At the present time L.V. is 2-5; R.V. 2-3; both unimproved by glasses. There is complete left ophthalmoplegia externa, with the exception of the external rectus and possibly the superior oblique. Ptosis is most marked when patient is not fixing. Pupils react very sluggishly to light and accommodation. With the ophthalmoscope few fundus changes are visible, but the outlines of the papilla are not clean cut—it has a woolly appearance; fields of vision in both eyes are decidedly contracted for white and colors. Dr. Sanger Brown reports that, with the exception of the oculo-motor paresis and optic defects, there is nothing abnormal about his nervous apparatus. Dr. Elliot found saliva and urine practically normal and free from lead.

ABRAHAMS.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 26th, 1897. President, Dr. Charles W. Burr, in the chair.

Dr. Judson Daland reported

A CASE OF DYSPHAGIA AND DYSPHASIA RESULTING FROM A LESION IN THE INTERNAL CAPSULE. (See this journal, page 614.)

Dr. Joseph Leidy, Jr.—This patient had been under my care for two or three years previous to his last attack. The symptoms referable to speech and to motor palsy entirely disappeared. His first attack occurred some eighteen months previously, and after an illness of a week or ten days, the motor symptoms, as well as the dysphagia and dysphasia, entirely disappeared. They were so evanescent that it was a question whether or not there was an organic lesion. He had recurring attacks after this at intervals of four or five months, and after each one the symptoms disappeared. Dr. Grayson examined the throat and found paresis of the right vocal cord. The speech was somewhat indistinct, but the tongue when protruded showed no deviation.

Dr. Wm. G. Spiller.—This is the second case of capsular monoplegia which has come under my observation. The first was a case presented by Ballet in his lectures in Paris, in 1895, but I am not aware that it has been published. The man, I believe, had paralysis of one limb, and at the autopsy a lesion was found in the internal capsule. I am under the impression that Dejerine has reported a case of capsular monoplegia, and that one has been recorded by one of the English writers, but I am unable to give the references to these. An important case of facial monoplegia of capsular origin was published last year by Etienne.

The disappearance of the symptoms in Dr. Daland's patient is hardly surprising, even if they were entirely due to a capsular lesion. Muscles which act together probably have a bilateral representation in the cortex, and certain of these muscles probably cannot be permanently paralyzed by an unilateral cortical or capsular lesion. Indeed, when the lesion occurs early in life, the restoration of function may be considerable, even when the pyramidal tract is destroyed, as in two cases with autopsy, which have recently come under my observation. In a

paper by Zacher a case is reported in which the hemiplegia in an adult only lasted a short time, with the exception of the persistence of a certain weakness and uncertainty of movement. At the autopsy, however, the corresponding pyramidal tract was found to be greatly degenerated.

It seems to me that the aneurysm of the right vertebral artery in Dr. Daland's case must be considered in explaining the symptoms. The paresis of the right vocal cord might have been due to pressure on the right vagus, and could not be explained by a lesion of the right internal capsule.

Dr. Judson Daland.—The case is interesting from the fact that we have here evidences of extreme arteriocalillary fibrosis with extreme cardiorenal changes. I am particularly interested in the condition of the right vertebral and basilar arteries. Careful examination shows that they are markedly dilated. It seems to me the dilated artery in this situation was abundantly able to exert compression on the pneumogastric, the hypoglossal, and probably the glossopharyngeal nerves. It may be that pressure on these nerves may explain a certain portion of the complex clinical picture presented by this case. I am also interested in the occurrence of Cheyne-Stokes breathing, a symptom which, when it occurs, is usually a speedy precursor of death. It seems to me that probably the chronic Cheyne-Stokes breathing was due in part to feeble circulation, in part to intermittent uremia, and in part to pressure exerted by the aneurysm of the right vertebral and basilar arteries.

Dr. A. Ferree Witmer presented a case of
ACUTE CHOREA IN A WOMAN AGED THIRTY-EIGHT.

This patient was a native of Scotland; was married; of temperate habits, and of excellent family history.

In the autumn of 1893 she fell a distance of twenty feet, but, aside from the shock, received no apparent injury, as she returned to her housework two days after the fall. Six months later the twitching began. This was sudden in onset, more marked on the left side, and has persisted with occasional weekly remissions to the present day.

The patient was of good physique; the eye grounds were normal; the vegetative functions and the mentality were undisturbed, and the speech was slow and of lowered pitch. The face, neck, body and extremities were the seat of abrupt, irregular movements. The tongue shared in these movements and gave rise at times to a clacking

sound as it darted to and from the teeth. The movements intermitted during brief volitional acts, but recurred later with increased violence. Rest lessened the intensity, but sleep did not restrain them. The gait was irregularly spasmodic; the knee-jerks were greatly exaggerated; and the elbow-jerks were present. The patient had been taking ascending doses of Fowler's solution for a long period without benefit.

In conclusion comparisons were made between this disorder and palmus, which in many features, the speaker thought, it resembled.

Dr. Wharton Sinkler.—This patient presents the usual picture of chronic, adult chorea, which may, or may not, be hereditary. Quite a number of patients in the nervous wards of the Philadelphia Hospital have presented very much the same features which we see in hereditary chorea, and yet there has been no history of similar trouble in their ancestors. The continuance of the movements during sleep is peculiar in this case, for in adult chorea, as well as in Sydenham's chorea, the movements do not continue during sleep.

Dr. Wharton Sinkler read a paper, entitled:

A CASE OF TROPHONEUROSIS OF THE HANDS (ACRO-TROPHONEUROSIS) WITH SPONTANEOUS AMPUTATION OF THE FINGERS. (To appear in a later number of this journal.)

Dr. Milton B. Hartzel.—I have observed this case for a number of years. I have been much interested in it chiefly on account of its great obscurity. It is singular that in a large proportion of these cases, the injury which preceded the gangrene has been a burn, not always by fire, but sometimes by chemicals. Until recently most of the cases reported have been in women. Two have been reported in men; in one of these the man is said to have been hysterical. I must confess that the term "trophoneurosis" does not convey much that is explanatory to me. In this case the burn occurred on the left arm, which is now healed. Why the trophic lesions should have developed on the right side is a matter that I cannot explain. So far as I can see, her condition is not at all changed, except that there has been great improvement in the general health. The condition of the skin is about the same that it was five or six years ago.

Dr. J. H. W. Rhein presented

A CASE OF ATAXIC PARAPLEGIA, AND A CASE OF
POISONING FROM BENZINE VAPOR.

Dr. Charles W. Burr.—It seems to me that the latter case is certainly largely, if not altogether, hysterical. Both history and symptoms point in that direction. The peculiar attacks which develop on fixation of an object certainly are not epileptic.

Dr. John K. Mitchell read a paper on

HEADACHE WITH VISUAL HALLUCINATION. (See this
journal, page 620.)

Dr. Joseph Leidy, Jr.—Has the blood in this case been examined?

Dr. Wharton Sinkler.—Does this patient have epileptiform attacks not in connection with the headache, and are these attacks induced by overfatigue, nervous excitement or similar causes? The visual hallucination is quite different in this case from those which usually occur in connection with migraine. In this case the long continuance of the visual hallucination, its definite form, and the conversation which the patient had with the giant would seem more like a psychical disturbance than a subjective visual disorder in the higher nerve centres. I have seen two patients who had visual hallucinations preceding epileptic attacks. In one case they took the form of a beautiful landscape, lasting for a moment, before unconsciousness, and in the other that of a hideous head.

Dr. William G. Spiller.—Scotomata, consisting of zigzag or fortification lines, are well known in ophthalmic migraine, but such hallucinations as Dr. Mitchell describes are, as far as I know, exceedingly rare. Cases of ophthalmic migraine with temporary hemiplegia, hemianesthesia and aphasia have been carefully observed. There are certain features of Dr. Mitchell's case, which are suggestive of hysteria, and if this affection can be excluded the case is a most valuable connecting link between epilepsy and migraine. It is well known that many neurologists believe there is a connection between these two diseases, at least between epilepsy and the ophthalmic form of migraine.

Dr. J. K. Mitchell.—I thought I had stated in my paper that no trace of malaria had been found. As to the occurrence of the attack, that, too, I thought I had made sufficiently clear. It never occurred except when the patient had headache; nor did it seem to be brought on by fatigue, emotion nor other

cause. If the case is one of hysteria, it certainly is a new variety of hysteria, and one which occurs only in summer time.

The point on which no one has commented is the one which seems to me the most interesting in the case—the apparent causal relation between the visual hallucination and the attack which followed.

Dr. A. A. Eshner read

A CONTRIBUTION TO THE STATISTICS OF THE MUSCULAR DYSTROPHIES.

He reported an analysis of twenty cases of progressive muscular dystrophy, collected from the records of the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases. These cases appear from the recorded notes to be of mixed type with a predominant tendency to pseudohypertrophy. Only four occurred in females, a proportion of 1 to 4. Gowers, in speaking of pseudohypertrophic paralysis, gives the proportion of the sexes as 1 to 4 to 7. In twenty-four cases of pseudohypertrophy, which came under his observation, he found the proportion 1 to 7 (three females, twenty-one males), and among two hundred and twenty cases, collected from various sources, the proportion was 1 to 6.3 (thirty females, a hundred and ninety males). In eighty-four cases Poole found eleven in females and seventy-three in males (1 to 6.6). Eichhorst quotes Seydel as having found among a hundred and twenty-five cases, twenty-two in girls and a hundred and three in boys (1 to 4.7).

So far as could be ascertained, the first symptoms in the cases here reported were noticed in none later than the thirteenth year; in two they were noticed at birth; in two during the first year; in two during the second year; in two during the third year; in two during the fourth year; in three during the sixth year; in one during the seventh year; in three during the eighth year; in two during the ninth year; and in one during the tenth year.

The ages of the patients at the time they came under observation were as follows: one was three years old; two, seven years; three, eight years; four, nine years; two, ten years; one, nineteen years; two, twenty-two years; one, twenty-four years.

In only two of the cases (brothers) was it definitely

stated that similar disease existed in other members of the same family, although in one other case the statement, which could not be verified, was made that a great-grandfather and a grandfather had suffered from a similar disease. These findings are not in accord with the results of the analysis made by Gowers of two hundred and twenty cases, of which a hundred and two were isolated, and a hundred and eighteen occurred in thirty-nine families. Erb gives the proportion of hereditary and familial cases as 56 per cent. In six of Poole's cases other members of the family had suffered from a similar disease.

In ten cases one or more of the infectious diseases had preceded the onset of the dystrophic manifestations. In two the labor was said to have been difficult; in a third, instruments had been used in the delivery; in a fourth, the mother had suffered from dystocia in a previous labor, on account of the large size of the child; and in a fifth, it was stated that the mother had suffered considerably from anxiety during the pregnancy. In three cases there had been falls upon the back before the advent of the symptoms of the muscular trouble. The father of one patient had been alcoholic and his family tuberculous; the father of another had died in the sequence of an apoplectic attack. An uncle of another patient had been insane. The grandfather of one had been paralyzed. One sister of another patient had died of meningitis, and one of another during dentition.

In two of the cases contraction of the muscles of the upper part of the thigh (in the one instance of the external muscles, in the other of the extensors of the leg) was noted when the sole of the corresponding foot was gently irritated, notwithstanding the absence of the plantar reflex. The writer has since observed a similar phenomenon under other conditions also, when the plantar reflex was not interfered with.

In none of the cases reported in this study is any specific reference made to the race of the patients, and the writer thinks it is not unfair to assume that even those cases in which there was no definite knowledge upon this point, occurred in white persons, inasmuch as the matter of race has always received special consideration at the Infirmary, and the physicians under whose care they were have no recollection of ever having seen a case in a colored person. Nowhere in the literature has the writer seen any

reference to the occurrence of the dystrophies in other than white persons, and his personal experience, together with information derived from a variety of sources, points to the rarity of these diseases in blacks. Numerous replies to letters addressed by him to neurologists and general practitioners in various parts of the country are in general quite confirmatory of the fact that the muscular dystrophies occur with the utmost rarity in colored persons, and especially in those of pure unmixed African parentage.

Dr. F. Savary Pearce reported

THREE CASES OF TRAUMATIC ANESTHESIA.

from the clinic of Dr. J. Madison Taylor, at the Howard Hospital. Two of these were the result of nerve section, and one of nerve contusion and compression. These cases showed entire loss of touch, pain and temperature senses in areas exactly defined by the distribution of the injured nerves. The speaker stated that in many instances where ill-defined areas of anesthesia exist after nerve trauma, all the fibres of the injured nerves are not divided, though in other cases complete severance is proven beyond doubt; and yet the loss of sensation or motion is not proportionate to the injury.

Case I. The patient, a colored laborer of thirty years, was referred from the surgical clinic of Dr. Edward Martin. Three weeks before coming to the hospital, he had thrust his right hand through a window pane and cut his wrist on the anterior side. A deep diagonal scar was found, two and a half inches long, just above the styloid process of the ulna, and extending over and through the median nerve. There had been, according to the statement of the patient, complete loss of sensation in the peripheral distribution of the median nerve since the second day of the injury. This loss of sensation was observed at the first visit of the patient to the clinic of Dr. Taylor. The thumb, index and middle fingers were edematous, and the index finger was ulcerated. The grasp of the hand was fairly good. Slight reaction of degeneration was found. Nerve suture was advised, and Dr. Martin united the divided ends of the nerve three weeks later. Restoration of function began at once, and the mobility of the hand was

covered *pari passu* with the sensation. The ulcer on the right index finger healed more slowly. The restoration of function was almost complete after seven months.

Case II. J. F., aged thirty, a laborer, fell from a scaffold five months previous to his visit to the hospital, and struck his right wrist on a piece of broken terra cotta ware, and probably completely severed the ulnar nerve just above the styloid process, inasmuch as all forms of sensation, according to the statement of the patient, were entirely lost a few days after the accident in the peripheral distribution of the ulnar nerve. This was the condition noted on his first visit to the hospital. Reaction of degeneration and dryness and brittleness of the nail of the little finger were noted. Suture was advised.

Case III. H. C., aged thirty-six, six days before coming to the hospital awoke after a debauch and noticed that his left eye was blackened, and that he could not feel on the left side of his nose and upper lip. The statement was made that on examination the anesthesia was found to involve the distribution of the left anterior dental nerve. The removal of a piece of bone in a depressed fracture at the left infraorbital foramen was advised, but the patient did not return.

GENERALYZED NEURO-FIBROMATA.

M. Delore (La Médecine Moderne, April 10th, 1897) reports a case of generalized neuro-fibromata in a child eleven years of age.

Three skin tumors appeared successively, one at the age of two years, on the right side of the nose; one at the age of five, on the left tendo-achillis; and the third at eight years of age, a little above the left breast. This was the largest of them all, and extended along the sixth left intercostal space. There was no spontaneous pain, but pressure, or contact with clothing, caused stabbing pains. On palpation there was a sensation as if touching a loose fibrous bundle with numerous nodules distributed upon it.

This neuro-plexiform tumor was removed by M. Vincent. It had two pedicles; one, external, consisting of thickened intercostal nerves; the other, internal, traversed the intercostal muscle, and was formed by an anterior branch of the perforating intercostal nerve. This growth was a fibroma of the nerve-sheath without alterations of the nerve fibres themselves. The other tumors were pure fibromas.

Periscope.

With the Assistance of the Following Collaborators:

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PSYCHOLOGY.

SUGGESTION AND PAIN. By C. M. Barrows. From "Proceedings of the Society for Psychical Research," Vol. XII., June, '96.

Mr. Barrow's stated that during the past seven years he has treated several hundred persons with "suggestion," who were suffering with various maladies. Among them he found the cases of pain to invariably succumb to the treatment. Here is one of his cases: A lady about to have a number of very sensitive teeth filled, inquired if suggestion would prevent the pain of the operation. He made no promise, but invited her to try. He explained his method and then suggested she would feel no pain while in the operator's chair that day. Two or three hours later in the day she was in the dentist's chair and remained for two hours without experiencing any pain, although she felt the grinding operation. The next day she called to report the success, instead of being prostrate in bed as on former similar occasions. At three other consecutive sittings (dental) her experience was the same, with a "suggestion" several hours prior to each appointment. But for her fifth appointment he (Barrows) neglected to give her the "formal suggestion," as he was in a hurry when she called, and as a consequence she suffered severe torture, and was prostrated for two days.

Each of his treatments consisted of but a simple suggestion, and he did not accompany the patient to the operator's office, nor continue the treatment after the patient departed. Each suggestion was a post-suggestion for a particular occasion and hour. Mr. Barrows mentions several cases of failure where the suggestion was not used at the precise time appointed. He treated seven cases of extraction of firm teeth with but one failure. One of the successful cases was on himself, having two molars removed, one being firm and one hooked, requiring the dentist to use two instruments for it. The case of failure was that of a woman who had four firm teeth and three loose ones removed.

The form of his therapeutic suggestion is silent, or telepathic, as he calls it. He says, "I use neither voice or other means to convey its import to the patient through sensory channels. I find it possible to affect, with these unvoiced suggestions, one who does not know my language, infants who have learned no language and brute creatures. This would be the case if communication depended on speech. More than this, I am not conscious of forming any statement of the mes-

sage, even in thought, when I make the suggestion. I certainly am not then thinking about my patient or at him. Using the term 'mind' in the popular sense, it does seem that the suggestions which I make are not addressed to it at all." "In all cases the mode of administration is essentially the same. When dealing with persons to whom it is new, I usually explain that the purpose of it is to render the nerves involved incapable, for the time being, of producing the sensation of pain. There is no hypnotism about this form of suggestion, no mesmerism, no animal magnetism, no mind cure. The effect does not interfere in any with the freedom of the patient, it works no charm and casts no spell upon him, exerts no control over his will, and makes no change in his thinking. My wish is to accomplish one definite result and nothing else, viz., to take away the feeling of pain from the consciousness which might otherwise suffer. If the patient is sceptical, I tell him that his doubt or faith will not alter the result, since it is not a matter of opinion but of sensation. Confidence in the treatment is not necessary, nor will any attempt to resist vitiate the effect. Having thus assured the patient and put him at his ease, I ask him to seat himself in a comfortable position, be quiet a few minutes, and think of anything he pleases, while I suggest to his nerves how to behave. Sometimes I show a patient what nerves are producing the sensation of pain, and locate the centres by placing my hand on his head. Some action of this sort is often useful with children, as a tangible evidence to them that something is being done." He presents the statement of a literary gentleman relative to his mental experience during the "treatments" he had received. He says: "I was never aware of the particular thing suggested each time, but only of the desired outcome of all the treatments. During the 'suggestion' we (operator and patient) sat quietly without speaking, my mind being perfectly at liberty. The suggestion occupied, as a rule, about twenty minutes, during which time I usually repeated poetry to myself or looked across to Boston, of which there is a fine view to be had from Mr. Barrows' windows."

CHRISTISON.

SOME MEMORY TESTS IN WHITES AND BLACKS. Psychological Review, May, 1897.

Geo. R. Stetson (Washington, D.C.) tested one thousand children in the fourth and fifth grades in the Washington schools, and found the colored children about equal to the whites in ability to memorize, a finding at variance with Ribot's idea that attention is a product of civilization. The colored children selected were the darkest in the schools. An intellectual difference between the two races was shown in the fact while the average age of the whites in the fourth grade was 10.63 years, and in the fifth grade it was 11.40, the average age of the blacks in the same grades was higher, being for the fourth grade 12 years and the fifth grade 13.14 years.

CHRISTISON.

DISCRIMINATION IN CUTANEOUS SENSATIONS. Psychological Review, May, 1897.

Leon M. Solomons (Harvard) experimented on two subjects with the points of a dull compass, sometimes using both points and sometimes one point about an equal number of times, but with a studied irregularity of order. They were applied to the "fleshy part of the forearm." One subject was regularly told he was right or wrong; the other subject was not. At the start both distinguished two points at about one and a half inches apart. After a few weeks' practice the subject who was regularly told he was right or wrong, had reduced the distance to about a quarter of an inch, while the perceptionness of the

other subject remained practically the same. This subject was then treated in the same way as the other, when he made rapid improvement. It was also observed that the ability to localize by requesting the subject to place his finger of the other hand upon the spots touched was not near so good as the power to discriminate between the sensations of one or two points. Also, it was found, that while simultaneous touching with two points required them to be one and one-half inches apart to produce the sense of doubleness, only one-half inch difference was required to produce the same effect by two points successively applied. Expectant attention was found to deceive every time. The conclusion is that the elements of number and space involved in the sensations are not cutaneous, but are the results of a mental process which is improved by training. CHRISTISON.

CLINICAL.

ALCOHOLISM IN NURSELINGS.

M. Vallin (*Jour. de Méd.*, Nov. 10th, 1896) believes that very injurious effects are commonly produced in infants by overindulgence in alcohol on the part of the wet nurses, and cites many instances in support of his opinion. Nearly all cases observed resembled each other. An infant several months old, will, without appreciable cause, become nervous, agitated and irascible. Offering the breast fails to calm it. During sleep it grinds the teeth, suffers from nightmare, or there may be complete insomnia. After these crises of agitation, a deep, prolonged, almost comatose sleep comes on, during which the face is distorted, and the limbs twitch with sudden jerking movements. Soon follow convulsions, sometimes feeble and localized, and sometimes general and almost incessant. After several days these symptoms disappear, to return at irregular intervals, probably in coincidence with new excesses on the part of the nurse. Vallin believes the majority of wet nurses drink at least one bottle of wine a day, and generally beer in addition. This amount he considers excessive, yet it is commonly exceeded. He would recommend the following as the proper allowance of liquid for a nurse: Half a litre of wine daily, taken while eating; one litre of milk, pure or diluted with water, during the intervals between meals, and fresh water at discretion, sweetened with some fruit syrup. FREEMAN.

TREMOR FOLLOWING LA GRIPPE. M. M. de Buck and de Moor. (*Jour. de Médecine*, Nov. 10th, 1896).

While the nervous manifestations consecutive to grippe are almost innumerable, tremor is of very rare occurrence in this connection, and few cases have been published. In the present instance the patient exhibited tremor, twice, after two attacks of influenza, a year apart, the second time much more marked than the first. The tremor involved the right arm and leg, was continuous while the patient was awake, but ceased during sleep. It was considered to belong to the category of hysterical tremor, being in a latent state until revealed by the grippe acting with predilection on the nervous system. The treatment consisted of subcutaneous injections of spermine. Every day 25 centigr. of hydrochlorate of spermine, dissolved in a generous solution of hydrochloral of sodium, were injected. After three injections a notable amelioration was observed. This treatment was continued for a fortnight, when the patient was able to resume his work. FREEMAN.

RABIES.

R. Lee Seward (*Virginia Medical Semi-Monthly*, Oct. 9th, 1896) reports a case of what seems to be a typical rabies. The patient was a negro, aged thirty-five, who had been severely bitten in the face by

an unknown dog, which he killed. The period of incubation was three months, and the duration of the disease four days. The man was not delirious at any time. Hypodermic injections of morphine and atropine seemed to decrease the severity of the spasms, but apparently did not delay the fatal termination. PATRICK (Chicago).

SCRIVENER'S PALSEY NOT SOLELY PEN FATIGUE. C. H. Hughes, M.D. (Alienist and Neurologist, XVII., Oct., 1896.)

This is a brief article setting forth the author's views upon "occupation" palsies. He denies that the occupation is the sole cause of the disease, and attributes it to a neuropathic organism that, by reason of excessive strain or overwork, becomes tired. The occupation is the determining factor, but to a very limited extent the predisposing cause of these neuroses. JELLIFFE.

NEURASTHENIC HÆMATEMESIS.

Dr. Ausset (Medical Week, Sept. 4th, 1896), reported at the French Congress of International Medicine repeated hæmatemesis in a patient with decided neurasthenic symptoms, not hysterical. The first attack was the result of overwork, the second followed a violent fit of anger. The possibility of tuberculosis, ulcer or cancer was carefully eliminated. MITCHELL.

ABSENCE OF CREMASTER REFLEX IN NEURASTHENIA.

M. Critzman stated his conclusions from an examination of nine cases of hereditary neurasthenia, to the Société de Biologie (Gazette Hebdomadaire de Médecine, July 30th, 1896). The cremaster reflex is almost never absent in normal individuals; it is absent in hereditary neurasthenics on both sides; it is also absent in persons having suffered with severe varicocele, and in those who have double orchitis. In acquired neurasthenia the reflex, so far as C——'s observation has extended, has been found normal. Finally there does not seem to be any connection in neurasthenia between loss of cremaster reflex and impotency. MITCHELL.

DES DEVIATIONS DU RACHIS EN NEUROPATHOLOGIE. (The deviations of the spinal column in neuropathology.) (Revue d'Orthopédie, 1896.) By Charles Mirallié.

The writer attempts to determine the frequency of deviations of the spinal column in nervous diseases. He depends on cases observed by himself and on those reported by others. A slight scoliosis may occasionally be found when hemiplegia has lasted a long time, and it seems to be the result of an attempt to remove the weight of the body from the paralyzed limb. It may also be observed in the infantile cerebral form of hemiplegia. The difference in the length of the limbs also aids in the production of scoliosis. Deviation of the spinal column has been found associated with idiocy, and in cerebellar hereditary ataxia.

Deviations of the vertebral column are common in anterior poliomyelitis of childhood, and usually develop late in the process. They are partly the result of unequal growth of the limbs, partly of atrophy and contracture of muscles, and of atrophy of the vertebræ. They occur also in the chronic form of anterior poliomyelitis, though rarely, and in Little's disease, and they may be an early sign of Friedreich's disease. Unless due to spontaneous fracture or atrophy of the vertebral column, they are not a sign of tabes. They occur rarely in multiple sclerosis, and are very common in syringomyelia, and are noted in Morvan's disease. Dejerine considers deviation of the spinal column a sign of great value in the disease described by him and Sot-

tas as interstitial, hypertrophic, and progressive neuritis of childhood. Scoliosis is very common in sciatica, and usually the convexity is toward the affected side. The position seems to be instinctively assumed in order to put the painful limb at rest. In some cases the convexity is toward the sound side, in other cases the convexity may alternate from one side to the other. Lumbar lordosis is not uncommon in progressive myopathy, and the peculiar gait observed in this disease has been compared to that of a duck (*démarche du canard*). Kyphosis has been observed in acromegaly as well as in the condition of osteo-arthropathy described by Marie, which closely resembles it.

Mirallié reports a case of hysterical scoliosis with cure. He thinks that in many of the cases of scoliosis of adolescence a history of some neuropathy in the ancestors may be obtained. SPILLER.

NEUROPATHOLOGY.

ASSOCIATION OF ACROMEGALY, EXOPHTHALMIC GOITRE, PHTHISIS AND GLYCOSURIA.

Medical journals frequently contain reports of cases of acromegaly, but the association of this disease with three other apparently diverse affections is so rare that it becomes of more than passing interest. An instance of this "remarkable combination" is recorded by Dr. George R. Murray in the *Edinburgh Medical Journal*, February, 1897.

Miss U., thirty-seven years old, single. At the age of twelve the thyroid gland became distinctly enlarged. From this time to the age of twenty she worked hard as teacher, and greatly suffered at night from a dull, aching pain in the left mastoid region. This pain gradually extended all over the head and became constant, while the thyroid gland increased in size, firmness and consistence. The expression of the face was fixed, denoting suffering of mind and body; forehead wrinkled transversely and vertically, and the supra-orbital regions were thickened. Eyeballs prominent, palpebral fissure narrow, upper eyelid drooping. The nose, lips and lower jaw typical of acromegaly. Most of the teeth are absent. Mastoid process large but normal in appearance. The thyroid gland quite large; the right lobe about the size of a hen's egg, the left nearly as big, while the isthmus was even larger than the right lobe. The spine of the seventh cervical vertebra was large and prominent, and there was a slight anterior curve of the spinal column in this region. The second rib thickened on each side, while the junction between the manubrium and the body of the sternum was marked by a prominent ridge. Skiagraphs of a hand and foot were taken with the following result: The total enlargement of the hand is due to an overgrowth of the soft tissues. The shafts of the phalanges are thickened, but the ends do not appear to be larger than normal. Neither the metacarpal bones nor the phalanges are increased in length. The middle and terminal phalanges of each finger, the (proximal) epiphysis is separated from the shaft by a narrow line, indicating that complete osseous union has not yet taken place between the two. The foot shows the increase in the soft structures, but the outlines of the bones are not well marked to indicate whether any change has taken place in them or not.

The skin soft and moist, and freely perspired. The pulse frequent, 120-140 beats in the minute—regular, soft and compressible. Cough and expectoration present. Examination of the chest revealed tubercular consolidation, with softening of the upper part of the upper lobe of the left lung. The urine clear, pale, acid in reaction; sp. gr. 1035. The single sample which was examined contained 40 grs. of sugar to the ounce.

The same writer reports another case of acromegaly, in a woman sixty-three years old. The additional and interesting features of this case were the large cystic goitre and constant spasm of the left face.

Excellent photographs, skiagraphic and pictorial, accompany the reports. In commenting on his cases, the author remarks: Only a few cases have as yet been examined by the help of the Röntgen X-rays, so that the changes revealed are of interest. The skiagraphs show the great increase in the bulk of the soft structures of the hands and feet, an increased thickness of the shaft of each of the phalanges of the hand, and the absence of osseous union between the phalanges and their epiphyses. As far as the author is aware, this greatly delayed union of these epiphyses has not been described before in acromegaly; this is rather surprising, in as much as this disease is characterized by an increased formation of bone. The author has so far recorded five cases in which acromegaly and exophthalmic goitre have occurred simultaneously. In four of them there has been glycosuria, and in two phthisis as well.

ABRAHAMS.

CONTRIBUTIONS TO THE CLINICAL AND PATHOLOGICAL STUDY OF ACROMEGALY.

Dr. E. Conini, in the *Archivio per de Scienge Mediche*, Vol. XX., fas. 4, 1896, reports three cases of acromegaly. In the first case the objective symptoms of acromegaly are pronounced, the only subjective symptom being a slight morning headache. In the second case there is disturbance of vision, insomnia, symptoms developed rapidly—disturbance of sensation about the arms. Marked improvement followed the use of thyroidine. In the third case muscular atrophy was present along with a peripheral neuritis. The autopsy on this case showed congestion of the pia; cerebral oedema; the pituitary gland enlarged so as to resemble a pigeon's egg, and softened; the posterior part of the sella turcica narrowed and deformed; normal size of the thyroid gland, weight gr. 100; no trace of persistence of the thymus gland; atrophy of the nucleus of the forearms and hands, which stood in contrast to the hypertrophy of the corresponding bones. Microscopical examination showed histological alterations in the hypophysis, in the thyroid, and a neuritis of the radial nerves.

RABIES.

Sweeny and Denny (*Northwestern Lancet*, 1896) report three cases of rabies. The first patient was a man of fifty-seven years. The period of incubation was about sixty days, and the first symptoms were a feeling of exhaustion, headache and pain in the back of the neck, followed by vague pains in the arm and shoulder. He was extremely nervous, and on examination (two days from the beginning) mentioned having received a bite from a stray cur about eight weeks previously. His face was flushed, and he complained of difficulty in swallowing water. "Taking the glass in his hand he held it shakingly, drew long breaths, finally holding his breath and swallowing it. There was a tremor of the hand and irritable respiration." Speech was husky, and words were momentarily forgotten. The urine had a specific gravity of 1035 and contained sugar. December 11th, two days later, he was delirious at times, talking to himself, but swallowed better. The pulse was hard and bounding, the urine had a specific gravity of 1030, and contained two grains of sugar to the ounce. December 19th it was noticed that the knee-jerks were absent, and that the grasp of the right hand was less than that of the left; The wrist tap was also diminished in the right side. December 22d the stupor had increased, and the next day the patient was rapidly failing; pupils were not contracted, there was some ptosis, external strabismus, slight

deviation of the tongue to the left, right facial paresis, general hyperæsthesia, tremor of the hands and body, and hiccough. Coma succeeded, and he died easily and quietly. There was no autopsy.

The diagnosis was meningitis, made from the symptoms and course of the disease, especially the ocular paralysis, supported by a family history of apoplexy and tuberculosis. Early in the attack the diagnosis might have been hysteria, if it had not been for the presence of fever. The diagnosis was changed to rabies some time afterward, in the light of the subsequent cases. Mention is made of a rare symptom noted, according to the authors, only by Trousseau, viz., a tendency of the patient to kiss those about him. The duration of the disease in this case (twelve days) was unusually long.

The second patient was twenty-one years old, the period of incubation about three or four months (the dates given in the paper are conflicting) and the first symptoms appeared gradually and were chiefly mental. The patient talked in a "rambling, foolish manner, was prone to causeless laughter, and seemed to be in a general state of exhilaration." After several weeks of such symptoms he began to complain of rheumatic pains in the right arm and shoulder, and at the same time became more excited, sleepless and restless, wandering about the house and talking excitedly on an infinite number of subjects. His temperature was 100 to 100.5, and the pulse 80 to 100. Difficulty in swallowing appeared and rapidly increased, and there was frequent spasm of the respiratory muscles. A couple of days later, "after a wakeful night," he became wildly maniacal, running up and down stairs, talking irrelevantly and incoherently, and spitting quantities of frothy and tenacious saliva upon the floor, walls and bed-clothes. His temperature was 102.5, and his pulse 120, full and bounding in character."

One of the authors gives the following description of the patient on the day before his death: "The patient was sitting up in bed, talking in a loud and excited manner, welcoming me effusively and stating that he felt extremely well. His face was flushed, eyes congested, pupils semi-dilated, and conjunctiva injected. Movements of the eyes, as well as accommodative reflexes, were normal. The lower lips trembled when at rest; the tongue protruded straight, but was thickly furred. The general expression was one of exhilaration and excitement. There was a marked difference in strength of grasp of the right hand as compared with the left, and he complained of numbness and pain in the hand and arm. The pulse was extremely full and bounding, its rate being 140, the temperature 103. The superficial, cremasteric, knee and ankle reflexes were normal, and there was no impairment of strength except of the right hand. Sensation was perfect everywhere. The patient, during the time that the examination was in progress, was talking incoherently, laughing, tossing about and spitting incessantly upon the walls and bed-clothing, but on being spoken to sharply he became rational and quiet. When I offered him a glass of water there was a general shuddering of the body as he took it in his hand. Then, after repeated urging, he attempted swallowing. A peculiar spasm of the respiration at once came on, that can best be described by the effort of a person to "catch his breath" after a sudden effusion with cold water. There was a rapid series of inspirations, accompanied by slight noise, followed by a gulp as he followed the water and threw the glass away from him. The endeavor seemed to exhaust him greatly, and there was a marked acceleration of the pulse. With a piece of orange he succeeded better, there being a less degree of difficulty and spasm. There was no fear of water, but intense dread of the spasms produced by the effort to swallow it."

The diagnosis rabies was only made after death, when careful in-

quiry elicited the fact that the patient had been bitten on the hand by the same dog that had bitten the first patient.

The post-mortem examination, made four days after death, was negative, excepting venous congestion of the internal organs and hyperæmia of the brain. The microscopic examination of the nervous system was also negative. A rabbit was inoculated, but the animal died at the end of four days from other causes.

Case 3. A man of twenty-six, began to complain of pain in the right arm and shoulder and side of the head, and to be restless and sleepless, ten or eleven weeks after being bitten on the right wrist by a small, stray dog. After about a week he was found to have slight fever at times, and complained of some difficulty in swallowing. During the next few days he became more restless, semi-delirious, did not sleep at all, the difficulty in deglutition increased, and he frequently ejected large mouthfuls of frothy, tenacious saliva. This was followed by a maniacal condition. "When asked to swallow some water, he took the glass in his hands, raised himself to a sitting position, and the lower lip twitched violently. As he approached the glass to his lips a violent spasm of respiration occurred, the patient taking a dozen rapid and shallow inspirations, accompanied by a grunting noise, and finally he poured the water into his mouth, swallowed it with a gulp, and lay back exhausted. The effort was accompanied by considerable increase in the pulse rate, and considerable distress of mind. His intellect, when his attention was attracted by questions, was perfectly clear, but the moment he was left alone his mind wandered off to his delusions again. He ejected frothy and tenacious saliva at times upon the bed-clothes and floor, preferring that method to swallowing it.

He rapidly became weaker, pulse 160, the face somewhat cyanotic, delirium marked, and he died comatose, having had no general convulsions.

The post-mortem examination, made twelve hours after death, showed a considerable excess of cerebro-spinal fluid, and a marked congestion of the blood vessels, which were prettily mapped out against the gray substance of the cortex. A rabbit was inoculated from the medulla, and it is said to have died with symptoms like those of inoculated rabies. Two individuals, bitten by either dogs, respectively, were treated at a "Pasteur Institute," and have shown no signs of the disease.

PATRICK (Chicago).

DOUBLE OPTIC NEURITIS IN TYPHOID FEVER. (Braine-Hartnell, Brit. Med. Jour., May 29th, 1897.)

The diagnosis between typhoid fever and meningitis, especially in children, is often exceedingly difficult, and the author relates an instructive case which calls attention to the fact, mentioned by few authors except Gowers, that choked disc may occur in typhoid fever.

The patient was a boy of eleven, who for eighteen days presented an anomalous group of symptoms, abdominal and cerebral, with fever, ranging from almost normal to 105.2 just before death. The symptoms and the course of the affection were such that it seemed impossible to make a positive diagnosis until two days before death, when inequality of the pupils, and well-marked double optic neuritis were noted, more pronounced upon the right side. From the presence of these symptoms the observer was strongly inclined to a diagnosis of meningitis, but the autopsy revealed distinct inflammation of Peyer's patches, with a decided enlargement of the mesenteric glands and solitary follicles. Examination of the brain and meninges was entirely negative.

PATRICK (Chicago).

Book Reviews.

CLINICAL LESSONS ON NERVOUS DISEASES. By S. Weir Mitchell, M.D., L.L.D., Edin. Lea Brothers & Co., Philadelphia and New York, 1897.

In examining a book with the above title from the pen of the distinguished author, one naturally expects to find a series of important observations, and his anticipations are realized. It is perhaps well, in reviewing this work, to select only a few of the interesting statements for special mention.

The first case is one of hysteria with psychic anesthesia for touch and psychic blindness. Mitchell says his patient was "mind-touchless." This is probably better than the name of tactile aphasia, for this case, at least. It might be well to limit this latter designation to those cases in which the patient recognizes an object but cannot give its proper name from touch alone. Failure to recognize an object and its uses by touch is more akin to mind blindness.

The author speaks of recurrent melancholia, and by this he does not mean circular insanity (*folie circulaire*), but refers to those cases in which the attack returns at a certain time of the year. He reports an extraordinary case in which the attack returns in March of each year, and lasts until August. There are certain very suggestive signs of hysteria in this patient. Several other cases of seasonal melancholia are mentioned. Unfortunately the writer can give no explanation of this tendency of the disease to return at definite periods. He speaks also of menstrual and intermenstrual melancholia, and raises the important question as to the beneficial effect on the disease of loss of blood. He describes also melancholia following dreams, and reports a case of a diabetic person in whom the attacks of melancholia seemed to be intimately connected with indigestion.

Mitchell speaks also of the hallucinations in the insane occurring during and even limited to the period he calls the *prædormitium*, and by this he means the period of gradual onset of slumber, when the mental powers are yielding to the gradual approach of unconsciousness. In the *postdormitium* similar sensory delusions may be present.

The condition of night palsy is a most interesting one, but since the publication of the recent excellent paper by Higier we must be prepared to accept some transitory paralyses as epileptic equivalents. The sleep ptosis, sleep pain, and sensory shocks, are not common conditions, and the latter are closely allied to the sudden jerkings of the body most of us have at times experienced while falling asleep; fortunately, however, few have been subject to the extreme forms described by Mitchell. The cases of chorea and tonic spasm, seen only on waking from sleep, are also extremely rare. We cannot help longing for a microscopic examination of the nerve cells in a case of the rare affection known as respiratory failure in sleep. This form is very suggestive of the asthenic bulbar paralysis.

The cases of subjective coldness without change in the objective temperature, especially when neuritis may be suspected, must remind

us of the pressure anesthesia with dissociation of sensation described by Biernacki.

The rapidity of the development of joint disorders—within four days, in some of Mitchell's cases—is almost proof positive that their origin is not in descending degeneration of the pyramidal tract and involvement of the cells of the anterior horns of the cord. This view has never been satisfactorily founded on autopsies, and the possibility of tertiary degeneration in hemiplegia has by no means been universally accepted, and certainly not of such degeneration within a few days. Darkschewitsch has written a valuable paper on these joint lesions.

Mitchell describes his treatment of sciatica by means of the bandage and splint, and in some cases by ice.

We are not surprised to find a chapter on erythromelalgia. The credit for our knowledge of this disease is universally given to Mitchell, and his name is usually mentioned in foreign journals in papers written on this subject.

The chapter on the wrong reference of sensations of pain is also an interesting one. This location of sensation in the limb opposite to the one irritated has been called, by Obersteiner, *alochiria*.

The chapters on pseudocyesis, hysterical contracture, and rotary movements, are valuable contributions to the neurological literature.

SPILLER.

INEBRIETY: Its Source, Prevention and Cure. By Charles Follen Palmer. Fleming H. Revell Company, New York, Chicago, Toronto, 1897.

This is a study of inebriety by a layman and written from the standpoint of the educator and moralist rather than of the physician. While the author is somewhat fanciful and diagrammatic in his analyses of the human character, the book on the whole is a very sensible contribution to the subject of the prevention and cure of inebriety. The author does not recommend specifics or Keeley-cures, but a systematic training in self-control and manliness and a cultivation of the general health and of a well-balanced body and mind. The book should serve as a sort of Bible for those who are inebriates or who have a tendency toward inebriety. It is unfortunate that the large percentage of those who have such tendencies and habits are the kind which will not read such books as this.

C. L. D.

THE
Journal
OF
Nervous and Mental Disease

AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-third Annual Meeting, held at St. John's Parish Hall
Washington, D.C., May 4th, 5th, and 6th, 1897.*

The President, Dr. M. A. Starr, in the chair.

Dr. John Jenks Thomas, of Boston, presented a paper, entitled:

TWO CASES OF ACUTE ASCENDING PARALYSIS,
WITH AUTOPSIES.

The first case was that of a woman, thirty-six years of age, who entered the Boston City Hospital, August 29th, 1896. She noticed a weakness of the legs, beginning suddenly on Aug. 5th, which became a complete paralysis the next day. The legs felt numb and dead, and were somewhat painful to pressure. There was retention of urine. On examination on entrance into the hospital the legs were paralyzed, except that the toes and feet could be moved slightly; the patellar and plantar reflexes were absent, and sensation was not affected. The arms were also paretic. There was no tenderness in the upper extremities. The internal organs were normal. The area of splenic dulness was not increased. Later there was bronchial breathing in the left back, and considerable dyspnea. The urine showed a trace of albumin and was neutral in reaction. The specific gravity was 1.018. A few questionable granular casts and a little blood were found in the sediment. The paralysis of the extremities increased, and there were incontinence of urine and dyspnea. The patient

died on August 24th. The temperature varied from 100.5 deg. to 102.0 deg. F. at first; later from 98.4 deg. to 100.0 deg.

The autopsy showed a bronchopneumonia of the left lung; chronic diffuse nephritis; arteriosclerosis of the aorta; myoma of the uterus, and an acute anterior poliomyelitis.

The cultures from the heart, liver and spleen were sterile. Those from the kidney gave colon bacillus and a liquefying organism, and those from the spinal cord also showed liquefying organisms (putrefactive organisms). The microscopic examination of the spinal cord showed, first, acute inflammatory exudation in the anterior horns of the gray matter, with parenchymatous degeneration of the nerve cells and processes; secondly, infiltration of the perivascular lymph spaces and dilatation of the vessels of the anterior horns; thirdly, moderate infiltration about the vessels of the posterior horns and of the white matter of the cord; fourthly, slight parenchymatous degeneration of the nerve fibres of the white matter of the cord; fifthly, slight degeneration of the posterior nerve roots and marked degeneration of the anterior nerve roots; sixthly, parenchymatous degeneration and perivascular infiltration of the peripheral nerves; seventhly, absence of microorganisms in the sections. The brain could not be obtained for examination.

The second case was of a man, aged thirty-five, who entered the Boston City Hospital on Oct. 5th, 1896. He had been well up to twelve days before entrance, at which time he began to complain of numbness and weakness in his legs. He had had very little pain, but was said to have had one attack of dyspnea before entrance. He ascribed his illness to exposure to cold and rain. On entrance there was marked paralysis of both legs, both arms and of the muscles of the neck. The tendon reflexes were lost. The plantar reflexes were present, but diminished. The cremasteric and abdominal reflexes were normal. Sensation was normal, and there was no tenderness. Urine was acid and without albumin and sugar; the sp. gr. was 1.025. The patient had an attack of dyspnea on August 7th, in which he died. The temperature was normal.

The autopsy showed bronchopneumonia, and hemorrhage of the lungs, enlargement of the spleen, congestion

of the kidneys, congestion of the brain and cord, parenchymatous degeneration of the peripheral nerves and of the motor cells of the spinal cord.

Cultures from the heart, liver, spleen, kidneys and meninges of brain and spinal cord were sterile. Four cultures were taken from the brain, three from the meninges of the brain, and one from the spinal cord. Three of these (two from the brain and one from the meninges) showed no growth. Dilution and replants from the others showed nothing but saprophytes (a spore-forming bacillus), a large coccus, and a large diplococcus.

The microscopical examination showed: first, parenchymatous degeneration of the peripheral nerves, varying in extent, and present to a greater or less degree in all the nerves examined; secondly, degenerative changes in all the large ganglion cells of the anterior horns of the cord, with destruction and fragmentation of the protoplasmic granules and loss of the nuclei of the cells; thirdly, the nerve cells of other portions of the gray matter of the cord, oblongata, brain and spinal ganglia unchanged; fourthly, no change of the white matter of the cord; fifthly, absence of micro-organisms in the tissues.

The author concludes that acute ascending paralysis is a clinical collection of symptoms arising from an affection of the primary motor neuron, probably toxic in nature (but not due to a distinct micro-organism), which varies in character in different cases, consisting in the milder forms of a degeneration of the motor cells and processes with disappearance of the chromophilic granules of the protoplasm and loss of the nucleus and nucleolus; and in the severer forms amounting to an acute inflammatory process of the anterior horns of the gray matter with infiltration of the perivascular lymph spaces and degeneration and destruction of the motor cells and processes.

REPORT OF THE COMMITTEE OF THE AMERICAN NEUROLOGICAL ASSOCIATION UPON THE AFTER-CARE OF THE INSANE.

This Committee was appointed upon the motion of Dr. C. L. Dana, of New York, at the meeting of this Association which was held in Washington, at the time of the last (1894) Congress of American Physicians and Surgeons. It was the outcome of a paper which was read and discussed at that meeting on the management of convalescence and the after-care of the insane.¹

The work of the Committee was begun by issuing a circular letter to certain prominent alienists and neurologists in the States of Massachusetts, New York and Pennsylvania. This number was afterwards increased in order to ascertain the sentiment of authorities on the subject in other parts of the country.

The letter ran as follows:

My dear Doctor:—

At a meeting of the American Neurological Association, held at the last Congress of Physicians and Surgeons in Washington, in 1894, a Committee on the After-care of the Insane, consisting of Dr. H. R. Stedman, of Boston; Dr. C. L. Dana, of New York; and Dr. F. X. Dercum, of Philadelphia, was appointed. Its purpose is to investigate and to report to the Association upon some feasible plan for the aid and supervision during the first month or two after their return from asylums to public life, of discharged pauper insane patients, who are recovered or improved.

Asylum physicians often hesitate, you are aware, to set at liberty certain patients whose condition seems to have so far improved as to make it useless to keep them longer under treatment, for fear that, thus thrown suddenly upon their own resources without oversight or perhaps means of support, they will fall back into the old habits of life which gave rise to their insanity. This applies also to patients who have recovered. These unfortunates are also distrusted and prevented from obtaining employment, simply because they have been inmates of an asylum.

These considerations led, in France, to the founding by Dr.

¹Journal of Nervous and Mental Disease, Dec., 1894.

Falret, in 1841, of an association for providing protection, assistance and homes for this class. It was, however, restricted to the department of the Seine. Its efficient operation has led to the recent establishment throughout that country, under the auspices of the French government, of *sociétés de patronage* (aid societies) for such discharged patients. Similar systems are in operation in England and Switzerland.

The office of an after-care society is to find for such discharged patients, according to their individual needs, suitable homes and places of employment; to provide gifts of money, clothing or tools; to redeem articles in pawn; to advance payment for rent, etc., etc., and finally to have them under supervision for the first month or two after their discharge.

We are of the opinion that the same need exists in this country, and that the work within our institutions for the insane should be supplemented by the same measures of outdoor relief on their discharge that have proved advantageous elsewhere. As this is an undertaking which has for its object the diminution of insanity by delaying or possibly preventing relapses, it seems to be called for both in the interest of humanity and of public economy.

The Committee would value your opinion on the subject and respectfully asks replies to the enclosed questions:

I. What are your views as to the practical utility of such an undertaking, generally speaking?

II. In your opinion should such an association be entirely a private charity, or would the co-operation of the State in its work be practicable?

III. Do you think it probable that benefit to a sufficient number of patients would result from the establishment of convalescent homes as departments of, but at a distance from our State hospitals for the insane? This inquiry is suggested by the proved usefulness of convalescent homes as adjuncts to general hospitals, and of summer cottages in connection with private institutions for the insane.

IV. (For superintendents of hospitals for the insane). Will you kindly give a rough estimate of the probable number of patients who have been discharged during the past year from the hospital under your charge, whom you would consider deserving of, and likely to be benefited, by such a charity, mentioning any special instances that may occur to you.

Yours very truly,

HENRY R. STEDMAN,
CHAS. L. DANA,
F. X. DERCUM,

Committee.

The result of this inquiry is as follows: Fifty replies were received; scarcely half a dozen less than the number of letters sent; a remarkable showing for a circular letter. Thirty of these are from superintendents of hospitals for the insane. They are for the most part full and comprehensive and, as might be expected from the practical experience of the writers, throw much light on the question. Thirteen only are from neurologists, as, in view of the apparently unanimous sentiment at the meeting in favor of the general adoption of after-care provisions for the insane, it seemed unnecessary to extend the canvass further in this direction. The remaining six replies are from members of boards of lunacy and charity.

Of the entire number of correspondents but six are either doubtful of the desirability and practicability of after-care associations for the dependent insane, or are decidedly opposed to such a step. The reasons given by them are that the number of cases which are likely to be benefited by such aid is too small to make it advisable; that while such a project might be desirable, it is inexpedient; that while excellent in theory it would be impossible in practice, etc. By far the most forcible argument on this side of the question will be found in the appended letter of Dr. Thos. G. Morton, Chairman of the Committee on Lunacy, of the Board of Lunacy and Charity of Pennsylvania.

On the other hand, the overwhelming majority of forty-four in fifty (or forty-seven in fifty-three, if the Committee be included) express, and in not a few instances, in the strongest terms, their decided belief in the great advantages to result from properly organized and conducted aid societies of this kind for discharged insane patients who are recovered, convalescent or improved. It is impossible within the limits of a committee report to give even a summary of the experience and arguments offered by these observers. The published replies, which are appended,¹ must be read to adequately appreciate the strength of the sentiment in this direction.

Regarding the number likely to be benefited in this way, the experience of superintendents of hospitals for the insane, as indicated in these replies, is not of much assistance, as the few who replied found it difficult to properly estimate the number of deserving patients. It would seem as though special inquiry of the individual patient when he leaves the hospital as to his circumstances and future surroundings is not customary in many institutions, although in some States he is provided with a sum of money or clothing. Fourteen only of those superintendents, who had charge of the class of patients to whom such a charity would be applicable, answer this question. Of these, nine think that there were "many" appropriate cases among patients who had been discharged from

¹ The letters will be published in the Transactions of the Association.

the hospitals with which they are or have been connected. Some of them say "great numbers" or "a great many," and one "nearly all discharged." Of the remaining five, three think that a few patients a year from each hospital might, perhaps, derive benefit from such an organization, while two think that no benefit at all would result.

As to the auspices under which such associations should be conducted, it is the general opinion of both the alienists and neurologists that they should by all means be begun as private organizations, and so continued until their utility be demonstrated. If successful, State aid could probably then be obtained.

In reply to the question regarding the advisability of establishing State homes for convalescent patients as a part of the general policy of States toward their insane, there is more diversity of opinion but the same degree of interest and careful consideration of the subject. Scarcely a member of this Association has written in opposition, and of twenty-nine hospital superintendents and other alienists nineteen were warmly in favor of such an accessory provision, five were doubtful of its expediency and five were decidedly opposed to the project. Of four members of lunacy and charity boards, one is in doubt as to its utility and the rest think it would be an unnecessary and useless experiment.

As the result, therefore, of their inquiries on the subject of the after-care of the insane, your Committee reports the following conclusions:

First. It is the general and well-nigh unanimous sentiment of those who are the most conversant with the needs of the insane in this country that measures should speedily be inaugurated for the temporary relief of discharged recovered convalescent and improved insane patients of the dependent class by organized outside assistance.

Second. As a preliminary step, inquiry should be made of all such patients before they leave the hospital, regarding the mode of life, surroundings and occupation to which they are returning, and appropriate advice given by a medical officer of the hospital. This precautionary measure is, we believe, too often neglected in large institutions for the insane.

Third. The legal provision, whereby an allowance of money and clothing is made in some States to each patient on his discharge, should be adopted by all.

Fourth. Outside assistance can best be provided, we believe, through the medium of an after-care association which, until its utility be proven, should be entirely a private undertaking, and should be organized like most existing charitable associations depending upon voluntary contributions. Obviously, a large city offers the best field for starting and developing such a system.

Fifth. The special methods of after-care relief by such an association should be those employed by similar organizations in other countries: England, France, Switzerland, or a selection of the best methods of each; these may be modified later to meet special conditions.

Such relief should, at first at least, be extended only to the class mentioned, and be understood as temporary, covering only the first month or two following the patient's discharge. The work may be best done by associates or agents appointed for the purpose, who shall find suitable homes and situations for all proper cases. There should also be systematic supervision of the homes by agents for the time specified or until the patient seems to be under good conditions for taking up life and work again. This applies also to patients returning to bad surroundings in their own homes. Reports should be made and records kept of each case.

Sixth. We believe it a duty that is especially incumbent upon this Association to take up in this way the work of the hospital physicians, and to see that the good accomplished in institutions be supplemented by proper outside supervision in appropriate cases; and we would urge its members to actively engage in the formation in their respective States, of relief associations for the after-care of insane patients of this class on their discharge from hospitals, and to endeavor to enlist in the work of co-operation all friends of the insane so far as practicable. To facilitate this, your Committee would suggest that a brief compilation from all available sources of the methods employed by such organizations abroad, be authorized and published by the Association for distribution to all who are interested in furthering this work.

Seventh. Regarding State convalescent homes, there is abundant evidence of the most authoritative kind of the advantages to follow from their establishment, but, in our opinion, the first reform in the order of precedence should be the general recognition of the necessity of separate hospital treatment of insanity in its early and active stage, and the actual adoption of special provision for the "acute" insane as an indispensable step in the hospital treatment of public insane patients. Only when this result is reached should separate establishments exclusively for convalescents be added to the already large burden of expense for our dependent insane.

The Committee acknowledge with gratitude the assistance given them by the numerous and full replies to their circular letter.

HENRY R. STEDMAN,
C. L. DANA,
F. X. DERCUM,

Committee.

Session of Thursday, May 6, 1897.

A REPORT OF TWO CASES OF BRAIN TUMOR.

By J. ARTHUR BOOTH, M.D.

The two cases which form the basis of this contribution to the general subject of intracranial growths, seem of some interest, and serve as an illustration of the division of the subject into two groups, viz., first, into that group in which the tumor is situated in some part of the cerebral axis or base, and therefore not to be reached by the knife; secondly, into that in which the tumor occupies some part of the motor area, in which class relief by surgical means is to be considered.

The first case in this report may be placed under the former heading, the history being as follows:

F. H., female, 28 years of age, single, came under observation on January 24th, 1894. She had always enjoyed good health, and had no trouble until five years ago when Dr. David Webster was consulted for a tired and strained feeling in the eyes. He found hypermetropic astigmatism, for which condition proper glasses were ordered. In April, 1893, the patient had a severe attack of the grippe, which was accompanied by acute and constant headache, being confined chiefly to the frontal region. This cephalalgia continued during the summer, becoming more general, and several times she vomited during the early morning hours. In the fall, on resuming her school work, she noticed a failure of vision, and on December 9th Dr. Webster was again consulted, and found double optic neuritis.

Examination—The patient is decidedly anemic, otherwise her appearance is that of health. Her equilibrium is poor, especially when she is standing on the right foot alone; she stands but a few seconds and then falls to the

left. There is no decided paralysis of the extremities. The grasp of the hands, as indicated on the dynamometer, in the right is 21, 19, 20; in the left, 23, 15, 23. The knee-jerk is decidedly higher in the left leg. On inspection, there is no paralysis of the face, but on testing the muscles the movements of the left cheek are a little more active than those of the right. The pupils are equal, and react to daylight. The reaction is slower in the right eye to a small ray of artificial light. There is no hemiopic inaction, but the angle of reaction is limited a good deal on all sides in both eyes. Her vision is 20/30 in each eye. The ophthalmoscope shows obliteration of the discs; the vessels are very tortuous. Numerous hemorrhages are also present. The patient describes peculiar illusions about the position of objects. She always sees the real object, but it is misplaced. In the last few days she has noticed a hemianopsia of varying type. While sitting opposite to two men in a street car, she saw only the man on her right side, or, as she puts it, she "saw one with her right eye, but could not see the other with her left." One day two different objects seemed to approach one another; for instance, the calendar on the wall moved over to the window curtain. When she looked at a friend who was reading to her, his left ear was over his left eye. The visual fields are normal. Pain, tactile and muscular senses are normal. There is no cardiac murmur, and the urine is normal.

Diagnosis—Tumor of the brain, and probably of the base; although no positive regional diagnosis was made. The patient was ordered increasing doses of potassium iodide.

February 20th. For the last two days the patient has suffered from vomiting and much prostration. Iodide of potassium has been stopped. The swelling of the optic discs is such that they can only be located by the general convergence of the vessels, and yet she has a vision of 20/30. Yesterday she fainted and thought she was going

to have an attack of paralysis; the left hand trembled and felt numb. She also complains of loss of smell. The tests show complete loss of the sense of smell for a white rose, elixir of valerianate of ammonium, putrid urine and cologne; chloroform is recognized as sweet, and ammonia is strongly felt. The anomalies of vision still continue. A horse that stood before the door, when looked at, seemed to contract so that he was made up of head and neck, and his legs and tail were so approximated that these parts seemed to form the whole horse. There is a slight paresis of the external rectus of the left eye. Her gait appears normal, though she says there is a tendency to stagger to the left.

March 14th. The vision is failing; in the right eye it is 20/50; in the left it is 20/70. She has had a severe headache in the occipital and suboccipital regions. Yesterday she had a fainting attack with loss of consciousness for a few seconds. The examination of hearing reveals a decided loss in the left ear. A watch is heard at a distance of fifteen inches in the left ear, and five feet in the right. The tuning fork applied to the glabella is heard best in the right ear.

April 16th. She had a sudden fall; both knees giving way. A few days later, while walking with her mother, she felt weak in the knees, and fell back heavily, striking the carpeted floor with the occiput. There was no swelling nor cut, and no nausea after the fall, but a severe occipital headache followed, and sight was much impaired. Since this attack there has been a rapid loss of vision, and now the patient has only a dim perception of light.

July 1st. All the symptoms have gradually become worse. The patient is now blind and confined to her bed. Within the last month a left hemiplegia, affecting also the face, has developed, with loss of sensation and paralysis of the right external rectus.

A great many notes were taken, but it is only important to state that the progress of the case was continually

downward. She gradually became stupid; lost her memory; sank into coma, from which she could be aroused at first, but which soon became profound, and in this condition she died, on January 29th, 1895, not quite sixteen months after the first occurrence of her headaches.

At the autopsy, twenty hours later, the head only was examined. Nothing abnormal was noticed in the calvarium. The depressions from the blood vessels and Pacchionian bodies were well marked. The membranes were much congested, and there was decided bulging over both parietal regions, but more marked on the right side. On section of the right parietal lobe a tumor mass was found in the right ventricle, completely filling it and extending over into the left. In consistence, the posterior portion was soft and broken down, while the anterior was quite firm. After hardening in Müller's fluid and alcohol, the following sections were made:

Left hemisphere—First section, longitudinal, was 4½ centimetres in the thickest part. Second section, longitudinal, was one centimetre in thickness. Third section, longitudinal, was one centimetre in thickness, and passed through the tumor.

Fig. I. The tumor, as seen from the left side in the



FIG. I.

median line, was pear-shaped, with the larger end pointing posteriorly, and invading the corpus callosum and fornix anteriorly, and occupying both ventricles. It measured 4 centimetres in its vertical diameter, and 6 centimetres antero-posteriorly.

Right hemisphere—First transverse section was 4 centimetres in thickness, and passed through the enormously dilated ventricle and cut off the anterior portion of the growth, which measured 1 centimetre in thickness. Second section was 3 centimetres in thickness, passed through about the middle of the ventricle and cut off a section of the tumor 3 centimetres in thickness. Third section was $2\frac{1}{2}$ centimetres in thickness, and exposed the mass completely. It involved a large part of the optic thalamus and corpus striatum. The tumor measured $7\frac{1}{2}$ centimetres on the right side; $6\frac{1}{2}$ on the left, and $5\frac{1}{2}$ centimetres transversely; the greatest vertical diameter being 4 centimetres. The microscopical examination, by Dr. Frank Ferguson, showed it to be a round cell sarcoma.

Case 2. Mary P——, 27 years of age, single, was referred to me by Dr. Giles, of the Manhattan Eye and Ear Hospital, on May 1st, 1896. She had always been delicate as a girl, and within recent years had been much troubled with headaches and nasal catarrh, the latter condition causing a loss of the sense of smell. The present trouble commenced two years ago, when she began to notice a blurring of vision after a short use of the eyes, especially in sewing or reading. The headaches increased in severity, became constant, and were localized in the eyes and occiput. Eighteen months ago she began to have epileptoid attacks, limited at first to the big toe of the right foot, but later extending so as to involve the leg and arm of the same side. There was no loss of consciousness in these attacks. Lately the right leg has become very weak; often causing her to stagger in walking, and this condition has been present for three months. The blurring of vision gradually increased, and in February last the patient no-

ticed that she could only distinguish light with the left eye. Her eyes were examined by Dr. Giles, who reports as follows: "Double optic neuritis; apparently receding in the left. Swelling of the right disc is 6 D.; of the left, 3 D. Vision in the right eye is 20/30; in the left, nil." For the last month the headache has been especially severe in the early morning, and is usually accompanied by vomiting without nausea. She complains of peculiar numb sensations in the whole of the right side, but careful examination does not reveal any sensory changes. There is no paralysis of the face or tongue, no aphasia nor any speech disturbance. Her equilibrium is poor, and she staggers in walking. The knee-jerk is increased, and is much higher on the right side. Ankle clonus is absent. The right leg is weaker than the left. The grasp of the hands is about equal; that of the right hand being 20, 20, 18; of the left, 20, 18, 20. The senses of hearing and taste are normal, while that of smell is lost.

Diagnosis—The presence of the following symptoms, viz., headache, morning vomiting, intense optic neuritis, paresis of the right leg, staggering gait, and epileptoid attacks, without loss of consciousness, were certainly sufficient to warrant the diagnosis of an intracranial tumor, and from the nature and character of the Jacksonian attacks, the situation of the growth was thought to be in the upper part of the left motor area, involving the centres for the foot and leg. The patient was also examined by Dr. Starr, and the above diagnosis confirmed.

Increasing doses of iodide of potassium were of no benefit, and on May 31st she was admitted to Roosevelt Hospital for operation. On June 2d, 4th and 7th, she had typical Jacksonian attacks, in which the convulsive movements were limited to the right side; the jerking beginning in the right foot and extending so as to involve the leg, thigh and arm, without loss of consciousness. These attacks lasted about a half hour, and were accompanied by severe headache and vomiting.

June 10th. The nurse observed that shaving and preparing the head for operation caused a twitching of the right foot and leg, and that there was marked tenderness over the vertex to the left of the median line. Prior to operation a slight elevation at this point was noticed. At 4 P.M., the patient being etherized, Dr. McBurney proceeded to operate.

A large oval flap, with its base towards the middle line, was made over the upper two-thirds of the fissure of Rolando. The veins of the scalp were enormously enlarged, and loss of blood at this time was considerable. The flap being dissected back as rapidly as possible, a small area, where the bone had been eroded, and a tumor-like mass projecting through it, were found. Pressure on this with a sponge caused convulsive movements of the right leg. The edge of bone was cut away all around with rongeurs for half an inch. Beyond this portion the bone was of normal thickness. An attempt was then made to remove the tumor, as it seemed flat and extradural, and this was done without any trouble. The dura beneath was very much thickened. After the bone had been cut away still more, the dura was opened, the limits of the tumor were revealed, and the finger was introduced in front of it. By means of traction with hook and finger it was lifted out of its bed. In the attempt to remove another piece of the growth from near the median line, a sudden gush of blood occurred, which was evidently from the superior longitudinal sinus. The bleeding was finally controlled by means of a pedicle clamp and sponge, compressing the sinus against the bone. The handles of the clamp were then tied together, and the instrument left in position, the dressing being applied around it. Notwithstanding hypodermatic stimulation and transfusion, the patient gradually sank, and died about four hours after the operation.

Pathological nature of the growth: The tumor consisted of three portions; the first situated on the dura, measured $1\frac{1}{2} \times 1 \times 1\frac{1}{4}$ centimetres; the second (Fig. II.), the



FIG. 11.

main one, rounded and lobulated, measured $3 \times 2\frac{1}{2} \times 2$ centimetres, was firm in consistence and weighed $3\frac{1}{2}$ ounces; the third, which lay near the longitudinal sinus, measured $1 \times \frac{1}{2} \times 1\frac{1}{2}$ centimetres. Microscopical examination showed the growth to be a sarcoma.

Dr. Philip Zenner, of Cincinnati, read a paper entitled:

SOME INTERESTING CASES OF BRAIN TUMOR
(to be published later).

REPORT OF A CASE OF SARCOMA OF THE BASAL GANGLIA, MID-BRAIN AND PONS, WITH SECONDARY ASYMMETRICAL HY- DROCEPHALUS.

By C. A. HERTER, M D.,

Visiting Physician to the City Hospital.

On Feb. 4th, 1897, an infant, nine weeks of age, with asymmetrical but well-developed hydrocephalus was admitted to the service of Dr. Reuel B. Kimball at the Babies' Hospital. The family history was entirely negative, the mother having eight other children, all of them healthy. The child was well nourished during the first few weeks of life, but soon ceased to nurse, and began rapidly to lose weight.

At the time of admission the child was $23\frac{1}{2}$ inches in length, with a circumference chest measure of 13 inches. The head measured $21\frac{1}{2}$ inches in circumference, $13\frac{1}{2}$ inches from ear to ear, $16\frac{1}{2}$ inches from nose to neck, and the right side of the head was very much larger than the left. The veins covering the head were markedly distended and tortuous. There was an interval of $3\frac{1}{2}$ inches between the frontal bones at their upper part, and a separation of $2\frac{1}{2}$ inches between the parietals. The child lay habitually in a position of pleurosthotonus, with the convexity to the right. There was constant rigidity in the extremities, the spasm being usually more pronounced in the arms than in the legs, and at times more marked on the right than on the left side. The pupils were small and responded feebly to light. Ophthalmoscopic examination by Dr. Arnold Knapp showed advanced atrophy of the optic nerves. The knee jerks were not obtainable.

The child was under observation in the hospital for

two weeks. During this time the circumference of the head increased nearly one inch. In other respects there was little change in the conditions noted above. From time to time the child uttered shrill cries. There was some disturbance of intestinal digestion, and occasional vomiting. At the time of death the child was nine weeks old. It is said that the head did not begin to enlarge until the child was three weeks of age. The enlargement of the head was thus very rapid.

Eighty-two hours post-mortem. Body—weight, 14 lbs. $2\frac{1}{2}$ oz. No skin lesions; poorly nourished. Some edema of feet and ankles. Rigor mortis fairly well marked. Brain—1300 c.c. of fluid removed. On removal of dura, convolutions everywhere very much flattened; pia normal in appearance; very little fluid between dura and pia; ventricles enormously distended with fluid.

Brain substance considerably softer than normal.

Occipital lobe, inferior surface; brain substance $\frac{1}{2}$ c.m. thick, temporo-sphenoidal, inferior surface 3 m.m., frontal lobe, inferior surface 2 m.m. Ependyma everywhere perfectly smooth, slightly reddened in places and covered by a considerable number of small radiating vessels, a little more prominent than normal. The distance between the outer borders of optic thalami measures $8\frac{1}{2}$ c.m. From anterior border of cerebellum to anterior extremity of corpus striatum, 7 c.m. on either side. On either side of basal ganglia apparently some induration and infiltration of the adjacent white matter.

Base—Optic chiasm is wholly indistinguishable, optic nerves very much thickened, and the entire central basal region protrudes downward and appears to be extensively infiltrated and enlarged. Pia covering peduncles apparently thickened. Coronal sections through the basal region, beginning with the anterior extremities of the thalami, indicate an infiltration of the entire central basal region with grayish material of rather firm consistence in places, in other places of rather gelatinous consistence.

A coronal section, 1 c.m., anterior to posterior end of pulvinar of thalamus, shows the third ventricle to have a transverse diameter of $1\frac{1}{2}$ c.m. and the same vertical diameter, the canal grows rapidly smaller from this point forward and is entirely obliterated by the tumor at a point about $1\frac{1}{2}$ c.m. anterior to level just mentioned.

Section at level of posterior end of pulvinar—Canal is blocked; there is infiltration and increase in consistence; indications of third nerve visible; fibres of third nerve, as they course from nucleus downward to point of exit, are more widely separated than normal; they are also more irregular in their course.

Section through pons—Near its upper border showed indications of same blocking of central canal. Entire pons on section infiltrated and much enlarged.

Measurements of pons—Vertically, 2 c.m., 8 m.m.; transversely, 2 c.m., 2 m.m. Central canal blocked, topography of pons only dimly recognizable owing to infiltration.

Floor of fourth ventricle presents a grayish appearance, especially in its lower half; the striæ acusticæ are visible, but not so distinct as normal, and the various contours corresponding to the cranial nerve nuclei are partially obliterated.

Anterior surface of pons retains its contour; pyramids are about normal in shape, but larger than they should be. Just below the calamus scriptorius upon the left side of the left restiform body is a small translucent mass about the size of a split pea.

Medulla is increased from side to side, apparently infiltrated by a gelatinous substance which extends $\frac{1}{2}$ c.m. into adjacent substance of the cervical cord.

Spinal cord.—In cervical region transverse maximum diameter of 12 m.m., anteroposteriorly, 6 m.m. The two lateral halves of cord are symmetrical. There appears to be a small fissure, about 1 m.m. in length, in right posterior horn in upper cervical region. In dorsal region there is a

wide fissure about 3 c.m. in length, extending into anterior surface of cord in median line; on either side of this, in the anterior horns, runs a vertical cavity about 1 m.m. in diameter.

Right Eye.—About the papilla are numerous small, irregular, sometimes oblong patches of pinkish tint, apparently disposed more or less radially to papilla. Left Eye.—Conditions less marked. Lungs—No pleurisy; right lung, posterior portion of upper and middle lobes consolidated; lower lobe solid. Left lung, lower surface of upper lobe shows strips of consolidation; lower lobe consolidated; on section, red broncho-pneumonia.

Bronchial lymph nodes normal.

Heart—Normal; foramen ovale closed.

Liver—Somewhat fatty. 5 oz. $1\frac{1}{2}$ dr.

Spleen—Normal. 3 dr.

Pancreas—Normal.

Kidneys—Normal. 1 oz. 3 dr.

Suprarenals—Normal.

Stomach—Normal.

Intestine—Ileum and jejunum normal.

Colon—Solitary follicles enlarged.

Mesenteric lymph-nodes—Normal.

Culture—(on glycerine agar) from hydrocephalic fluid showed bacillus coli communis (probably post-mortem contamination).

Microscopic Diagnosis:—Spindle-celled sarcoma of the basal region of the brain with small metastatic tumor near restiform body. Double optic atrophy. Broncho-pneumonia with purulent infiltration. Fatty liver. Parenchymatous degeneration of kidneys.

Microscopical examination. Tumor at base:—Coronal sections through mid-brain, beginning anteriorly, show infiltration of the substance with large spindle cells which have very granular nuclei; scattered among these cells are found also some small and some large round cells.

Spindle cells lie side by side forming strands which run

in every direction, forming in some areas a feltwork in the meshes of which are groups of spindle and round cells.

In some areas the cells are closely packed giving the stroma a dense appearance; in other areas the meshwork of cells is looser.

Stroma moderately vascular. Sections through middle and posterior portions of tumor show gradually decreasing infiltration of the brain substance.

Sections including portions of the margin of the circular aperture in centre of tumor mass, whose gross appearance gave indications of being the dilated third ventricle, show this margin to have a ragged edge; no indication of inflammation found; in one place well-preserved columnar epithelial cells are visible for some distance; they are evidently the epithelial cells of the ependyma.

Sections stained with picro-acid fuchsine show more distinctly than those stained with eosin and hæmatoxylin, what appears to be an intimate connection between the adventitia of the blood vessels at one point and the most densely infiltrated portion of the tissue at another; fine strands composed of spindle cells run from the vessels for a considerable distance, finally appearing to blend with the cells of the most markedly infiltrated areas.

Pons—Much less infiltration of the substance with spindle-shaped and round cells. Central canal considerably dilated; epithelial lining well preserved. Outlines of ganglion cells indistinct; nuclei pale.

Medulla—No infiltration of substance; nerve cells better preserved than those in pons. Portion of choroid plexus included in section, shows grouped about one or two of the larger blood vessels aggregations of spindle cells which appear here as above to spring from the adventitia. The small metastatic tumor external to left restiform body shows same structure as stroma of large tumor at base, save that the structure is quite loose, cells being separated from each other as though by fluid (gross appearance was one of translucency).

In several places the fibres of the pia seem to be growing into stroma of the tumor.

Cervical enlargement—A breaking down of gray substance in both anterior horns to form small cavities; canal patent; epithelial lining intact.

Mid-cervical region—Anterior fissure wider than normal; passing downward it increases in breadth, and in dorsal region it gapes widely.

Cavities in anterior horns exist until mid-dorsal region is reached, where they cease. In lower dorsal region a deep fissure is seen, extending from anterior surface of cord through right anterior column to a point nearly on a level with the spinal canal; canal in this region contains granular material.

Optic nerves show advanced atrophy of the nerve fibres.

Lungs—Epithelial lining of bronchi almost completely destroyed; many bronchi clogged with pus cells and desquamating epithelial cells. Walls of bronchi are intensely vascular and infiltrated in many places with small round cells. Alveolar walls show thickening, and their capillaries are much congested; contents of alveoli; desquamated respiratory epithelium; pus cells and pigment-carrying leucocytes; this pigment is deposited also in the interstitial connective tissue. Some of the broncho-pneumonic areas show very marked purulent infiltration.

Liver—Shows advanced stage of fatty degeneration; most of the parenchyma cells converted into fat, but here and there are small groups of epithelial cells fairly well preserved, with palely staining nuclei.

Spleen—Some distention of the cavernous veins.

Pancreas—Normal.

Suprarenals—In places fatty degeneration of the epithelial cells of the cortex.

Kidneys—Granular degeneration of the epithelial lining of the secreting tubes. Contents, fibrin and cell fragments. Loops—Epithelium shows slight degeneration.

Collecting tubes—Epithelial cells swollen, nuclei pale, in few places cells breaking down. Vessels—Normal. Tufts—Capsules normal, except in one or two places where they partake of a degenerative change described below. Epithelium of capsule swollen and desquamating. Capillaries congested; tufts in most instances completely fill capsules. In several places the tufts show a degeneration somewhat resembling amyloid change; the degenerated material is homogeneous in appearance, staining well with eosin, and gradually replacing the tuft. In a few instances Bowman's capsule is involved in this degenerative change.

Besides the very rapid increase in the size of the head, which was noted in this case, the hydrocephalus bore the exceptional character of being much more developed upon one side than upon the other. In observations relating to many cases of hydrocephalus in children, I have never met with a degree of assymetry equal to that noted here, and Dr. Holt, whose experience is larger, was equally impressed with the exceptional degree of asymmetry. Dr. Peterson, however, tells me that he has once or twice observed a marked inequality of the head in cases of apparently primary hydrocephalus.

In a case reported by Demme, the enlargement of the head, which became great, was apparently confined to the right half of the head and was noticeable at birth. Here there is no reason to suppose that the hydrocephalus was secondary to a new growth, for focal symptoms were absent, the optic papillæ were normal, and the child was still under observation when nearly four years of age, when the intelligence was good and considerable language had been acquired. The evidence thus favors the view that a primary hydrocephalus may not merely present marked asymmetry, but may be in very rare instances strictly unilateral. I am quite unable to think of a satisfactory explanation of the manner in which such cases arise.

In the present case the asymmetry of the hydrocephalus appears to depend upon the mechanical influence of the

neoplasm and its most reasonable explanation seems to be that the communication of the right ventricle with the ventricular system, through the foramen of Munro, was blocked at an earlier period than the corresponding communication of the left ventricle, both foramina having been eventually blocked by the infiltration of the new growth.

Microscopically the new growth presents nothing of interest except the relation of certain cells to the blood vessels, which makes it likely that the neoplasm had its origin in the connective tissue of certain vessels of the optic thalami, and that it arose from several foci at about the same period rather than from a single focus.

The appearances described as relating to the spinal cord impress me as artefacts.

In looking through the literature relating to rapidly growing sarcomata of the basal region of the brain in infants, I have not been able to find cases which bear a sufficiently close relation to the present case to make a comparative study of much interest.

I wish to express my indebtedness to Dr. Louise Cordes for aid in the preparation of the pathological report.

Dr. Angell:—I was very much interested in Dr. Herter's case because I have had a similar, though by no means so marked a case. In my patient the hydrocephalus was secondary to the pressure caused by the tumor, which was a cerebellar growth. We have not, fortunately for the child, succeeded in getting a necropsy, because the child still lives. While the enlargement of the head was not very great—I think the greatest circumference of the head of the child, at five years of age, was twenty-two inches—the side on which the growth was situated was decidedly larger. I do not think Dr. Mills, who saw the child later, nor I, advanced any theory to account for this. In this case we did succeed in getting a quiescent state of the growth under the influence of iodide of potassium and cod liver oil, although there was no specific hereditary history. The child now walks with the aid of a little chair, and there is some improvement in his mental condition.

Dr. Edward D. Fisher:—The cases Dr. Zenner has reported are of very great interest in that they show irregular types of brain tumor. Two or three cases have been under my own ob-

servation in which the usual symptoms of brain tumor have not been present; that is, cases in which the paralysis has not been marked, even although, as we found at the autopsy, the motor areas have been compressed. There has also been an absence of optic neuritis in these cases, and on the presence of this sign we naturally count a great deal in forming the diagnosis of tumor.

Dr. Zenner's statement, in reference to the relief observed in many cases from operation without removal of the tumor, I think is correct, and it is not necessary that we should operate at the site of the tumor to obtain this relief. A patient under my observation illustrated this point very well. In this case the tumor was found at the necropsy to be connected with the cerebellum. The operation had been done over the forehead and had given complete relief for three months, until the death of the patient from other causes. The convulsions with which the patient had been afflicted had entirely ceased. I have not always found percussion of the skull of great value in the localization of a tumor. It seems to me to be of service at times, but is not to be relied on, and is only an aid in forming a diagnosis.

In the slowly-growing tumors we find that the displacement in the motor areas is often so gradual as not to result in paralysis. I have seen this condition lately in two cases, in each of which a very large tumor was present, affecting the motor area of the brain and causing localized epileptic convulsions. In one of these cases also there had been no optic neuritis, and yet the localizing symptom of an epileptic seizure had been distinct. There was a very large tumor, about two and a half by three and a half inches, involving the motor areas.

In another case, which did not come to operation, a tumor was found at the autopsy depressing and distorting the pons. Paralysis had been noted on the same side as the tumor. Whether or not that will turn out later to be a case of the sort Dr. Zenner referred to, in which the pyramidal tracts do not cross, I do not know.

There are many cases of tumor which cause very few symptoms and last a long time, and the forming of a diagnosis in these cases, except from the general symptoms, is very difficult.

Dr. Pershing:—A man of middle age had general convulsions and loss of consciousness about two years before he consulted me. Since that time he had noticed a defect in vision, particularly while reading, and had found it difficult to follow the lines. There had been occasional attacks of convulsions, but almost no headache. Examination revealed a sharply defined quadrant hemianopsia, the upper quadrant of the right sides being missing in both fields. The optic discs were perfectly normal.

The apparently sudden onset led to a diagnosis of vascular

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occlusion, and the quadrant hemianopsia was attributed to softening in the region of the calcarine fissure.

After a time the patient grew rapidly worse. Headache then came on and was severe. The right side became paretic. Word-deafness and paraphasia were noted. The patient at one time asked his wife to put some ice-water on the fire. Within two weeks after the development of these symptoms he became comatose and died. There was no optic neuritis at any time. At the necropsy a firm tumor, about the size of a hen's egg, was found in the left temporo-sphenoidal lobe. Around this tumor, especially in the thalamus and crus, was a soft, infiltrating growth, which was evidently a glioma. The macroscopic appearance of the occipital cortex was perfectly normal. The hemianopsia must have been caused by disease of the pulvinar.

Dr. Langdon:—A case seen recently by me bears on the favorable results of operation referred to by Dr. Zenner. A man of fifty had stupor, right hemiparesis, paraphasia, right hemianopsia, subnormal rectal temperature for ten days, which only twice went slightly above normal, and a pulse of forty-five to forty-eight. It was finally decided to explore posteriorly to the left motor area. At the operation nothing was found but an anemic condition of the cortex. No pus was revealed by probing. Within forty-eight hours the man's mental condition improved, his pulse went from forty-five to sixty, his temperature rose to 98.4-99, and was not again subnormal. His improvement still continues, although the right motor paralysis is now absolute.

[Note.—June 16th—A necropsy obtained since the above remarks were made revealed an inflammatory mass (gumma?) subcortically situated in the left angular gyrus, and also extensive discoloration of the adjacent white matter. The angular gyrus was necrotic.]

A case observed by me in which the symptoms and the location of the growth indicated a non-decussation of the pyramids, was published in full in *Brain* in 1895. In this case the symptoms had existed for seven years. There had been double optic neuritis and all the evidences of an intracranial growth, which I was inclined to locate in the pons on the right side above the decussation of the pyramids. At the autopsy, however, the tumor was found on the left side, involving the pons and cerebellum above the motor decussation, and the pyramidal decussation was absolutely perfect. The motor and sensory weakness, however, had been on the same side as the tumor. The specimen was shown before the Neurological Society of London and caused considerable discussion. Two or three explanations were offered for the location of the growth on the same side as the symptoms.

Dr. Fry:—I recently saw a necropsy in which a diagnosis

of tumor of the cerebellum was confirmed. There was marked asymmetry of the skull. One lateral ventricle was more distended than the other. The foramen of Monro was open.

During life there had been a decided difference in the percussion note of the two sides of the skull. The percussion sound in this case, and in two other children with distended skulls from tumors of the cerebellum, has recently interested me. In all the cases percussion gave a peculiar cracked-pot sound, due, I suppose, to the wide separation of the bones at the sutures. I have not examined the literature sufficiently to see what has been said about this sound by others.

Dr. Hugh T. Patrick, of Chicago:—It seems to me that by far the majority of the cases of tumor we see are of the irregular type; each case of tumor has to be decided for itself on its own merits. I think that Dr. Zenner's former belief regarding the general convulsion preceding the local convulsion in deep-seated tumors, is correct. I have come to that conclusion, partly from experience, partly from reading a few reported cases, and, I suppose, partly from reasoning. I have been accustomed to be guided by this sign in questions regarding operations, of course with exceptions; and I have even been guilty of teaching this opinion.

I should like Dr. Zenner to speak a little more definitely. He says he found exceptions. I should like to know whether they were not really exceptions, and whether the rule does not hold good, and hence is really of value in diagnosing. I think it is, and that the primary convulsion in the deeply-seated tumors, which start within and grow toward the cortex, is apt to be general and the others local, possibly with general convulsions.

As to the incision in the brain, I feel inclined to advise it; always with due respect to the surgeon and his technique. If the operation is begun for tumor, and if we feel reasonably certain that it is there, it seems to me that the proper thing is to keep on cutting after the surface of the brain is reached, because we do have these typical symptoms from the subcortical as well as from the cortical growths. If we have sufficient symptoms indicating a focal origin, I believe, ever with deference to the judgment of the surgeon, that it is proper to go below the surface for the tumor, if it is not found on the surface.

As to Dr. Pershing's remarks, I think that the careful examination of the visual fields, and the careful examination for speech defects, are often of assistance in making a diagnosis and in localizing the tumor. These symptoms are sometimes searched for too superficially. Slight defects of speech are sometimes of real diagnostic assistance in the early period of growth of brain tumors.

Dr. W. M. Leszynsky:—I saw a patient recently in whom

there were symptoms of cerebral abscess complicating middle ear disease. Trephining over the temporo-sphenoidal lobe was performed and the parts fruitlessly explored. Within a few days the optic neuritis subsided and there was considerable improvement in the symptoms. The patient died, however, on the sixth day.

The President:—I have seen seven cases of brain tumor since the last meeting of the Society, four of which were operated on and the other three of which came to autopsy. I have seen in all, if you will grant my diagnosis, fifteen other cases; but these seven I know were tumor, because I saw the tumor. The percussion note was distinctly altered on the side of the tumor in three, and in the other four it was not.

In the first case, operated on by Dr. Weir, the tumor, a small infiltrating gliosarcoma, was found at the autopsy lying in the white matter of the apex of the temporal lobe on the left side. It was about an inch in diameter in all directions and quite vascular. For three months before the operation there had been a progressive motor aphasia, attended by paralysis, first of the right side of the face and then of the right arm; and (while the patient was in the hospital) of the right leg. The progression of symptoms is interesting. The aphasia was the first symptom—purely motor, not sensory; then there were twitchings of the right side of the face; then of the right arm; then paralysis of the right side of the face; then paralysis of the right arm; then complete hemiplegia. There were optic neuritis and also headache, but no general convulsions. It seemed perfectly clear that the tumor must be in Broca's centre. At the operation nothing was found, excepting a distended hemisphere, and no palpable tumor anywhere. Of course, there was no relief, excepting from the headache, and death occurred about ten days after the operation. This tumor was found at the necropsy with some difficulty, because the necropsy was made through the small opening of the skull, and the tumor lay at some distance below the portion of the brain exposed.

In the second case the patient was seen at the clinic, and had been under observation for about two months before we decided to operate. The general symptoms were very marked; they were optic neuritis, headache, percussion note very markedly different, great tenderness of the skull, and very slight hemiplegia. There were a number of general convulsions, and then the patient had a typical unilateral convulsion limited to the right side. We took that, of course, as the indication, and although she was in quite a comfortable condition and easily able to walk around the ward of the hospital the day she was operated on, yet we thought, inasmuch as the blindness was very marked, we had better operate. At the operation by Dr. McBurney we were much astonished, on exposing the motor area of the cortex, to find an enormous infiltrating gliosar-

coma measuring three and a half inches in one direction and two inches in the other, occupying the entire motor area of the brain—and yet the woman had walked around the ward as well as possible. The removal of the tumor was attended by enormous hemorrhage, and the day following the woman went into collapse and died.

The third case was that of a patient with all the symptoms of a brain tumor, which was easily located in the cerebellar fossa. Optic neuritis, vomiting and other symptoms were quite well marked, and the cerebellar staggering was characteristic. Nothing had been retained in the stomach for three weeks; the agony from the headache was awful; the man was nearly blind and insisted on something being done. Dr. McCosh operated, although we stated to the family that there was very little hope. The operation was done over the cerebellum, on the side on which it was pretty evident from cranial nerve affection that the tumor lay. The cerebellum was well exposed and appeared to be perfectly normal. The operation was, of course, a disappointment: Dr. McCosh thought something further ought to be done in the way of exploration, and he enlarged the opening upward in the hope that the tumor might be found. Not finding it there, he tapped the lateral ventricle, and we obtained immediately forty cubic centimetres of fluid. At the end of the tapping a drainage tube was placed in the ventricle and the patient removed to bed. The headache and vomiting immediately ceased. The man began to recover, and was very comfortable for the next two weeks, being entirely free from the general symptoms of brain tumor; viz.: headache, vomiting and general restlessness. There was marked relief of the general symptoms by this tapping of the ventricle. At the necropsy the tumor was found in the cerebellum, exactly under the place we had exposed, but it was covered by a perfectly normal layer of cerebellum. We could not have found the tumor during life; even at the necropsy it was difficult to distinguish its limits.

The fourth was the case of a little girl with symptoms of cerebellar tumor. These had come on very rapidly during three months, and then were absolutely stationary for about seven months. She was brought to my clinic. The child was totally blind and had headache and was unable to walk. We decided to operate and Dr. McCosh made the attempt. Our intention was to expose the cerebellum on the left side, because the cranial nerve symptoms were greater on the left side, and a flap of the skull was turned down, exposing the occipital bone. This was found to be full of small holes, through which the venous blood gushed. It was impossible to operate any further without causing death on the table. Dr. Walton, of Boston, has reported one case of a similar condition, where apparently an erosion of the occipital bone had occurred. It seemed to me

that this tumor had gone on producing symptoms during the first three months; had eroded the occipital bone; then that a collateral circulation had been established between the inside of the skull and the scalp, through these holes in the bone, and that this was the reason why there was relief in the symptoms which had become stationary for seven months.

I am impressed with the fact that so many of our symptoms of brain tumor are due to effusion into the lateral ventricles, and if that does not occur we are misled as to our diagnosis.

In another case, the man had most irregular symptoms, very largely of the mental type, occasionally attacks of hemiplegia, lasting for a few hours and then passing off entirely, also attacks of coma coming on gradually and passing off gradually. At the necropsy an enormous tumor was found in the right temporosphenoidal lobe, involving also the basal ganglia. There was little or no effusion into the ventricles, and the general symptoms in this case were few and slight.

As to asymmetry in hydrocephalus, I have never seen it. I reported to this Society seven years ago a tumor which looked very much like the tumor which Dr. Herter has reported, and photographs of that can be found in my article in Dercum's Text Book on Nervous Diseases.

Dr. Herter:—An interesting question is whether we are to regard a marked degree of asymmetry in hydrocephalus as being presumptive evidence that we have to deal with tumor. From what Dr. Peterson has told me I should judge that this is not the case, and I should judge also from the case of tumor that I referred to this morning, that this is not the case. At the same time it may be that most cases of hydrocephalus that present a high degree of asymmetry are due to tumor. If that is so, it is a point of interest in helping us to make a diagnosis in these cases. I judge that these cases of asymmetry are comparatively rare. In the literature I have come across several cases which resemble the one referred to here this morning, in which there was a tumor of the cerebellum, and in which there was a certain degree of asymmetry in the hydrocephalus.

Dr. Zenner:—In regard to the diagnosis of cortical and subcortical tumors, Dr. Patrick spoke of the character of the first attack. It is difficult to find many cases that directly point to this issue. There are cases of subcortical tumor where, according to the report, the first attack was of a Jacksonian type, and other cases where the first attack was of a general character. The same is true of cortical tumors. I had merely given up that point as of no value, although I thought subsequently that it is just possible that the reports have not been accurate in this respect. The case reported by the President is also against the view. I should have been glad to have heard more about the value of percussion of the skull in forming the diagnosis.

Another point was mentioned by Dr. Fisher. I saw a case a few days ago in which I suspected the existence of a tumor on account of the symptoms, the chief motor manifestations being slight ataxic movements of the right hand. I saw a case of that kind once before, where I did not have an opportunity to see the necropsy, but in which the existence of a tumor was undoubted. The question is in regard to the localizing significance of such ataxia.

The President:—As to percussion note, I have been percutting all the heads that I could get hold of for the last two years, and I think that it is not a reliable symptom. I do not think we can put a very large diagnostic value upon it at present. That is the reason why I mentioned that out of seven cases of tumor, the percussion note was changed on the side of the tumor in three, and not changed in the other four. I can add to that five cases of abscess of the brain, observed at the New York Eye Infirmary, and the percussion note was changed on the side of the abscess in three and not changed in two. It is a symptom that is irregular. As to the location of the tumor being cortical or subcortical, I do not think it affords any indication whatever. The change of percussion note is not indicative of cortical as distinguished from subcortical tumor.

As to ataxia, I have not found it at all common as a symptom in tumors, and I have been very much surprised that this is so. I have observed two cases, however, that are of interest, in which there was no ataxia prior to the operation, but in which a very marked ataxia of the hand developed subsequently to operation on the posterior central convolution. An angioma was removed by Dr. McCosh from the posterior central convolution of a boy who, although perfectly able to move before the operation, yet for three weeks after the operation was extremely ataxic, so much so that he could not feed himself with his right hand. I had the same experience this year in a case of epilepsy. A cyst was found and removed by Dr. McBurney. Subsequently to removal of that cyst an ataxic condition developed. After three weeks the symptom passed off. I have not seen it as a permanent symptom in tumors.

Dr. Joseph Collins and Dr. G. E. Brewer read by title a paper on the

REMOVAL OF PART OF A SUBCORTICAL CEREBELLAR TUMOR; DEATH TWO AND ONE-HALF MONTHS LATER.

The symptoms in the case were severe headache of acute development, and at first located chiefly in the back

part of the head, dizziness, vomiting, uncertain gait, with a tendency to go toward the left, slight uncertainty in the movements of the upper extremities, loss of the knee-jerks at a later stage of the disease, and choked discs, more marked on the left side and developing rather late in the process. There were no symptoms of sensory nor motor palsy, and the gait was not typically cerebellar in type, but rather that of asthenia. The patient, in an advanced stage of the disease, was unable to stand. On account of the absence of any symptoms other than the classical ones of cerebellar tumor, a subcortical growth in the substance of the right cerebellar hemisphere was suspected.

An omega-shaped incision was made, with the convexity upward, beginning in the median line opposite the rudimentary spine of the first cervical vertebra, extending upward and to the left, then arching over and toward the right mastoid process, then downward to a point about two inches to the right of the point of the original incision. Nothing abnormal was seen in the right half of the cerebellum, and an incision half an inch in depth was made into the cerebellar tissue. As nothing pathologic was found, a second and deeper incision was made near the median lobe, and the finger, at the depth of one and one-half inches below the surface, came in contact with a distinct nodule, about three-fourths of an inch in diameter. This was removed. The patient rallied and gained thirty pounds of flesh.

About two months after this operation it was thought best to re-open the skull on account of a return of the symptoms. Lumbar puncture was first performed. The patient died during the operation. A large tumor was found at the necropsy in the right cerebellar hemisphere, and proved on examination to be a typical caseous tuberculoma. Evidences of tuberculosis were found in other parts of the body.

(To be continued)

A CASE OF TROPHONEUROSIS OF THE HANDS
(ACROTROPHONEUROSIS), WITH SPON-
TANEOUS AMPUTATION OF THE FINGERS.¹

By WHARTON SINKLER, M.D.

The case which I have the honor to present to you this evening, was under the care of Dr. Louis A. Duhring for several years, and has been reported by him in the International Medical Magazine, March, 1892. I desire to acknowledge here my indebtedness to this paper for the early history of the case, and also for some of the references which I have used in this report. Dr. Duhring recognized from the beginning the neurotic origin of the disease, and he kindly placed the patient in my hands some months ago, with the view of seeing what benefit could be derived from a course of treatment directed to the general nervous system. The history is as follows: A. A., single, aged thirty-five years, was born in Philadelphia, and has had no occupation except assisting in housework. She is of small stature, frail and thin. There is no history of neuroses in the family, except that the father had a paralytic stroke at the age of sixty-one twelve years ago, which has materially affected his speech and memory. A younger brother is delicate and has a weak heart. For years the patient has had many symptoms of general nervous disorder, which included frequent and protracted attacks of gagging and vomiting, palpitation of the heart, crying spells, and globus hystericus. She lost flesh, suffered from insomnia, and had continued indigestion. Eighteen years ago she had an attack of chronic otitis media, which lasted four years, but left no permanent injury. She first menstruated at

¹Read before the Philadelphia Neurological Society, April 26th, 1897.

the age of fifteen years, but the periods were always irregular, and ceased completely about six months before the beginning of the present trouble, when she was twenty-seven years of age, and did not reappear until February, 1897. In September, 1890, she was burned with a flat iron on the flexor surface of the left forearm, just above the wrist, the area being about the size of a silver dollar. The burn was superficial, and only slightly blistered, and presented no peculiarities. It did not, however, heal readily nor completely, and from some unknown cause began to break out anew, and, within a month of the accident, began to show a superficial gangrenous patch which remained about six weeks. The patch began to be now more inflamed and painful, with at first darting and then aching pain, which continued in about the same severity for about two months. The whole forearm was reddened, swollen and tender, and was the seat of **throbbing** and darting pains. About six weeks **after** the accident the burn seemed to be nearly **healed**, and then a single pimple, a papulo-vesicle, formed on the extensor surface of the forearm, **near** the burn. In a week or two this lesion ulcerated and crusted, and then other similar pimples formed near the original one, some of them vesicles and some blebs, covering by degrees the greater portion of the wrist. Some healed, while new ones formed, most of them leaving scars as they passed away. Various forms of local treatment were employed, but none seemed to influence favorably the progress of the eruption. Arsenic was on several occasions prescribed, but had to be discontinued on account of intolerance of the drug. Every few weeks, or even oftener, an attack of swelling and painful inflammation of the arm occurred. These attacks were accompanied by severe pain. The pain was usually worse in severe or damp weather. The patient was first seen by Dr. Duhring, January, 1891, and he gives the following description of the local appearance of the disease: "The eruption consisted of small, irregularly-shaped, ill-defined,

chronically inflamed, vesicular and bullous, herpetic-looking, more or less crusted, scarred patch, with scars extending considerably beyond the inflamed skin. Apart from the old scars, it possessed at first sight the general appearance of an injury, due rather to the local action of an acid, or to some chemical substance, than to disease from within. There was, moreover, some oozing and discharge from the broken and ruptured vesicles, blebs and excoriated surfaces. The inflammatory process of the skin was superficial, for there was but little thickening, and the scars were not deep. The vesicular and bullous lesions were irregular in outline, more or less angular; distended, but with no disposition to rupture; some flat, others raised, and usually were unaccompanied by areolæ. They possessed an herpetic aspect, and from this characteristic formation were manifestly due to direct nerve influence. The crusts were depressed, saucer-shaped, and adherent to the skin in the centre, with everted edges. They were variegated in color, with bluish and blackish tints. The scars were plainly the result of the vesicular and bullous lesions. The patch was irritable, sore to the touch, and painful, and the whole extremity up to the shoulders was likewise the seat of darting nerve pain." The treatment adopted at this time was the use of a weak galvanic current to the nerve-trunks, but after a month's application no positive results were obtained. The diseased process continued on the left wrist and upper part of the hand for three years, migrating from place to place, and breaking out anew as soon as any points became healed; finally the condition of the arm began to improve, and by the early part of 1894 the left arm was entirely healed. Just at the time of the healing of the left arm, there appeared at the end of the right index finger a papulo-vesicle, similar to those which had invaded the left arm. This followed the same course of breaking out into an ulcer and then healing, new papulo-vesicles formed on this finger, and then involved the adjoining fingers. The affection sub-

sequently spread to the back and palmar surface of the hand, and finally involved all of the fingers. At times there appeared gangrenous patches on the fingers, which would be followed by sloughing, and more or less loss of tissue. In this way the first joint of the third finger was lost, and after a time the first joint of the index finger sloughed away. The patient continued in poor general health; she was very nervous, ate and slept but little, had frequent attacks of vomiting, and became, to some extent, addicted to the use of paregoric for the relief of the burning and boring pain which was more or less constant in the right hand. She was admitted to the Orthopædic Hospital and Infirmary for Nervous Diseases, October 23, 1896. The patient's general health was very poor; she was anemic, thin, and presented a general neurasthenic aspect. Her tongue was coated, and the digestion was bad. The left wrist on both aspects showed cicatrices which resembled those from the results of a burn. The mark of the original burn was distinctly seen on the inner side of the forearm. The skin of the right hand was coarse and seemed to lack vitality. The epidermis was loose and could be peeled off in strips. The last two phalanges of the index and ring fingers were absent, and the stumps were pointed and covered with a black leathery slough. Beneath this were granulations which discharged a rather offensive serum. There were many vesicles covering the back of the hand and the fingers; the middle finger was flexed upon itself and had no motion. The thumb and middle finger appeared to be healthy as regards the skin, but were somewhat atrophied, and the thumb was adducted so that the carpophalangeal joint was very prominent. At this point a supernumerary digit had been amputated several years ago. Sensation was unimpaired. The recognition of heat and cold was somewhat blunted in the stumps of the index and ring fingers, but there was no change in the thermal sense in the remainder of the hand and arm. Tactile sense and thermal sense in the left

arm and hand were normal. The urine was examined and found to be free from albumin or sugar. The patient complained of constant pain in the right hand; it was of a burning character most of the time, but sometimes there was a dull aching, and at other times lancinating pain; occasionally there was itching, which was annoying. She slept badly on account of the pain, and had a poor appetite. Her weight was seventy-five pounds, and her height was five feet. She was very anemic, and she had not menstruated for six years. The treatment was directed to the general neurasthenic condition. The patient was kept absolutely at rest in bed; given massage and electricity daily; visitors were excluded, and all communication with the outside world was cut off. The patient's general health improved markedly and rapidly. She gained in flesh and color, and on February 11, 1897, she menstruated for the first time in six years; but there has been no appearance of the period since that time. Her general condition improved so much that on March 28, 1897, she weighed a hundred and six and a half pounds, a gain of thirty-one and a half pounds since her admission. Her color was good, and her health was excellent. The condition of the hand varied greatly from time to time. There have been times when the nutrition of the skin has improved to such an extent that the skin has been entirely healed, as shown by accompanying photographs. After remaining well for a few days, there has been reappearance of the vesicles and bullæ, and accompanying these, there has been marked swelling and redness of the whole hand, and the appearance of a black slough upon the extremities of the fingers. The little finger, which had not been affected when the patient was admitted, has been attacked on two or three occasions, and sloughs have formed which have caused distinct loss of tissue. The beginning of the sloughing process is, first, discoloration of an area of skin involving, perhaps, one-half of the surface of the finger. This rapidly becomes black, then dries, and the slough is thrown off, leaving a granulating surface.

The patient is suffering at the present time from a fresh outbreak of the eruption in the right hand. One week ago the hand had entirely healed; then without any apparent cause, there was increase in the pain, the whole dorsal surface of the hand became inflamed, and a number of vesicles formed, which soon coalesced, so that the entire epidermis of the back of the hand was loose. At the same time, a large patch of discoloration appeared on the outer surface of the little finger, which in two or three days became black and gangrenous. A number of small points of discoloration also appeared on the back of the hand, which became black and gangrenous. The whole hand was swollen and inflamed, and the pain has been so



great that the patient has been unable to sleep at night. The only explanation of the present attack that I can suggest is, that the patient has been allowed to do more for the past two or three weeks than at any time since her admission to the hospital; she has been out of bed the greater part of the day; has been seeing her friends, and has had more opportunity for mental activity. She has not shown any evidences, however, of unusual nervousness.

The case is one of great rarity and unusual interest, and although a number of somewhat similar cases have been recorded, I have been unable to find in literature any which present the same features. There is no question but that the affection is a trophoneurosis, dependent upon

malnutrition of the nervous centers. It resembles in some of its features, Raynaud's disease, and yet in other respects it is totally different from this affection. The areas of gangrene which occur are not preceded by pallor and contraction of the vessels, and it is, therefore, not to be regarded as a vasomotor neurosis. Cases of similar character have been described as occurring in hysterical women, and they have been considered by some writers as being the result of hysteria. It is well known that certain extraordinary lesions of the skin occur in patients suffering from hysteria. Weir Mitchell has described a case in which the leg between the knee and the ankle was the seat of a skin



disease, in which a thick and dense crust formed, which resembled the bark of a tree. In a case of hysteria under my care, both legs, from the knee to the ankle, were the seat of an extensive vascular eruption, which had been preceded by profuse local sweating. Kaposi, of Vienna, reports a case of a female nurse, in whom a wound, which had been caused by a rusty nail, was followed by an eruption of blebs and areas of painful inflammation in the vicinity of the wound. He calls the disease "pemphigus neuroticus traumaticus hystericus." A number of other cases have been reported, in which a wound or a burn has been followed by a skin lesion, characterized by vesicles, blebs

and gangrenous patches. Some writers regard the affection as hysterical, and others regard it as dependent upon an ascending neuritis. Lancereaux (*Le Bulletin Médicale*, Oct. 7, 1891), describes a condition which he calls "herpetism." He regards this as being due to an individual predisposition of the affection. It is characterized by erythematous, papular, and vesicular eruptions. Sometimes there are marked evidences of articular rheumatism with deformity of the joints. The same author has recently written a paper on "Trophoneurosis of the Extremities" (*La Semaine Médicale*, 1894, page 261), in which he describes several forms of disease in which spontaneous amputation occurs, and refers again to the condition which he has described as "herpetism." He relates cases in which trophic disturbances closely resembled those of leprosy, resulting in loss of substance or the spontaneous amputation of one or more fingers. In most of the cases of trophoneurosis described by Lancereaux, in which there was spontaneous amputation of the fingers, to which he gives the name of "Autocopic Trophoneurosis;" the disease occurred either in old persons where there was arterial obliteration, or from a condition allied to ainhum. In one case which he describes, the patient, a young girl of twenty-two years of age, was born with but two fingers on the right hand; the other three had been amputated through the body of the middle phalanges, at the age of three or four years; the middle finger was amputated by a process of circular amputation at the second joint, and the great toe of the left foot was also nearly amputated by a furrow, which made its appearance at about the age of fifteen years. At the lower end of the right thigh there were also two semi-circular furrows, which made their appearance when she was about twenty-one years old, and which had gradually grown deeper. There was also in this case the appearance of some skin lesions in the form of purpuric patches.

In my patient the question of leprosy presented itself, although the lesion of the skin did not resemble this disease

in any respect, but the destructive process by which the greater part of the two fingers had been lost, made the question arise. Dr. D. Braden Kyle has very kindly made a careful examination of the blood, and of the secretions from the ulcerated surfaces, as well as of the serous discharge from the vesicles, and has given me the following report:—

“Blood and Inoculation from Hand—Red corpuscles, per cubic millimeter, total, 3,900,000; red corpuscles, per cubic millimeter, normal, 2,000,000; white corpuscles, per cubic millimeter, 35,000; hæmoglobin, 70 per cent.

“The red cells were very irregular, some of which were nucleated—myelocytes,—some crenated and corrugated. The first examination showed peculiar bodies in the red cells, but after repeated examinations of later specimens, no such bodies could be noted. It is possible that the bodies were granular areas in the red cell, which had a peculiar affinity for the blue stain. The erythrocytes showed a poor affinity for the acid stain. A few normoblasts were present. The white cells showed evidence of degenerative change, many being ruptured. The handling of the specimen would not account for such marked alteration. There were also free nuclei present. The lymphocytes were largely increased in size and numbers.

“Inoculation.—Tube inoculations showed a mixed growth, there being present bacilli and cocci. The cultures were plated, but by the second generation nothing but cocci could be found. These macroscopically and microscopically were staphylococci, and by testing on animals proved to be ordinary pus cocci, giving a negative result as to the presence of any special germ.”

The possibility of the existence of diabetes was also considered, but a careful examination of the urine showed that there was no sugar present. The absence of sensory changes or of thermal anesthesia excludes the possibility of the existence of syringomyelia. It is not likely that the disease could depend upon an ascending neuritis, due to

peripheral injury, because the disease which began in the left arm as the result of a burn, healed completely and permanently before the beginning of the trouble in the right hand. In the right arm there has been no tenderness along the nerve trunks, and none of the usual signs of neuritis. We are, therefore, forced to the conclusion that the disease is a trophoneurosis, dependent upon an hysterical diathesis. This conclusion is strengthened by the fact that the eruption healed completely when the patient's general health was improved, to a degree of excellence greater than it had attained for many years.

(For discussion of this paper see page 639.)

A CASE OF GENERAL MOTOR NEURITIS WITH ANASARCA. (Revue de Médecine, No. 1, 1897.) By J. Dejerine and C. Mirallié.

Edema is not very uncommon in peripheral neuritis, resulting from injury of nerves, but is usually localized, and is not extensive. It is much less frequently seen in neuritis of internal origin, and when it occurs does not affect a large part of the body. Dejerine and Mirallié, however, are able to report a case of general edema.

The patient, a man of fifty years, had an attack of bronchitis, angina, coryza and headache, with excessive lassitude. When seen two months later, considerable edema, almost complete, with paralysis was observed in the lower limbs. The paralysis of the upper extremities was of less degree, but, nevertheless, very evident. The face and neck were not affected. There was no loss of sensation, and there were no spontaneous pains, although the thighs and calves were very sensitive on pressure, and the pain was chiefly felt along the course of the nerves. The knee-jerks were abolished. The vesical and anal sphincters performed their functions normally. Later edema was observed in the upper limbs. There was also atrophy of the lower limbs, and the electrical reactions were altered, but there was no reaction of degeneration. Albumin was not found in the urine. The patient had tachycardia. As the edema disappeared the amount of urine became excessive. Recovery was complete. The writers think the case was one of peripheral neuritis, and that the edema was not due either to heart or kidney disease. They have only found the reports of two cases of anasarca in peripheral neuritis. The edema, they think, was due to paralysis of the vasomotor nerves. It was not a case of beriberi, although in some respects it resembled this disease. SPILLER.

HEREDITARY SPASTIC PARAPLEGIA.¹

By WESTON D. BAYLEY, M.D.

IN the family about to be described, it is known that five generations have presented cases of primary lateral sclerosis. How much more remotely this singular malady extended it is impossible to learn.

The genealogical tree here shown represents the one accessible branch of the family as far back as can be authentically traced. Other members of the fourth generation and their descendants are known to have been afflicted in like manner to the one here described, but no authentic information from them is at this writing obtainable. For assistance in the collection of these records, I am indebted to my friend, Dr. F. Mortimer Lawrence, who with me made personal examination of the appended cases.

The numericals on the diagram indicate the cases personally examined, and identify them in the records which follow.

H. F., unmarried, female, æt. 31, housewife (Case 1 of tree). Apparently well until five years of age, when she gradually became paraplegic, showing at first a spasticity of the adductors of the thigh. The trouble has been symmetrical from the start. At no time has there been pain, numbness or other sensory symptoms; no bowel or bladder difficulty, no atrophy, no static ataxia, no diplopia, no deformity of the feet. Muscles hold the limbs rigid. Knee-jerks are apparently absent, even with re-inforcement. Pupils equal—both react to light, but the right a little more promptly than the left.

C. F., married, male, æt. 28, telegraph operator (Case 2 of tree). Apparently well until five years of age,

¹ Read before the A. R. Thomas Club, of Philadelphia.

		Male unaffected.	
		Female " "	
	Male unaffected.	Male " "	
		Female " "	
		Male " "	
		Female " "	
	Female unaffected.	Female unaffected.	Female unaffected.
		" " "	Female unaffected.
		" " "	Female unaffected.
	Female afflicted (unmarried).	Female afflicted. 1.	Male afflicted. 9.
		Male " 2.	Male unaffected. 2 years.
		Female (died in convulsions act. 3 weeks.	
	Male afflicted (died act. 55 of apoplexy).	Male unaffected.	Male unaffected.
		Female afflicted. 3.	
		Male " 4.	
		Female unaffected.	
		Male afflicted. 5.	
	Male afflicted.		
	At least one afflicted brother of whom no definite information is at present obtainable. Descendants of him or his brothers or sisters are known to be afflicted.	Male unaffected (died in infancy of hip joint disease).	
Female afflicted.		Male unaffected.	
		Female " "	
		Male " "	
		Male " "	
		Female " "	
		Male " "	
		Female (died in infancy).	
		Female " "	
	Female unaffected.	Female (neurotic) unaffected.	Female under 3 yrs. unaffected.
		Female unaffected.	
		Female unaffected (neurotic, choreic etc.).	Female under 3 yrs. unaffected.
		Female (died act. 9 months of sp. meningitis.	
	Male afflicted. 6.	Male unaffected.	
		Female afflicted. 7.	
		Male " 8.	
		Female unaffected.	Female unaffected.
		Male " "	Female unaffected.
		Male " "	
		Female (died 1 yr. of measles.	
	Male unaffected.	Female unaffected.	
		Male " "	
		Male " "	
		Male " "	

when his "legs began to give out." The first symptom appeared to be hyper-adduction of the thighs, and later general spasticity of both lower extremities. This condition has persisted without material change to the present time. Paretic spastic gait with contractures of the tendo-

achillis. Knee-jerks are exaggerated, pupillary reflexes normal and very free, ankle-clonus on both sides. Has never had pain or numbness; no static ataxia; bowel, bladder and sexual reflexes are normal.

E. F., unmarried, female, æt. 19, housewife (Case 3 of tree). Apparently well until between her fifth and sixth year, when adductor spasm, soon followed by spastic paraplegia, developed. No pain or numbness. Knee-jerks very excessive, the slightest touch producing a vigorous response. Well-marked ankle-clonus in the left foot only. No diplopia; pupils react normally; no bowel or bladder symptoms; no static ataxia.

T. F., unmarried, male, æt. 16, schoolboy (Case 4 of tree). No illness until the fifth year, when spasticity of the legs developed, with adductor spasm without sensory symptoms. Knee-jerks excessive, marked ankle-clonus, no static ataxia, talipes equinus. Pupils equal and react normally. No bladder or bowel symptoms.

C. F., male, æt. 11, schoolboy (Case 5 of tree). No symptoms until his third year, when the spastic gait appeared in the same manner as with the others. Talipes equino-varus; knee-jerks free, slight ankle-clonus in both feet; the deformity has been progressive during the past year. Pupils equal, and react normally, no static ataxia, no bowel or bladder symptoms, no sensory symptoms.

C. F., married, male, æt. 51 (Case 6 of tree). Was apparently well until between his fourth and fifth year, when spasticity of gait appeared, being most marked at first in the adductors of the thigh. There developed early tendo-achillis contracture, and because of the deformity, tenotomy was performed twenty-three years ago. Since this operation he has not been able to walk nearly so well as before it. The knee-jerks are very excessive; no ankle-clonus because of the tenotomy. No sensory symptoms; no bowel or bladder difficulty. Pupils react normally; no static ataxia.

The two following cases are children of Case 6.

M. F., female, æt. 13, schoolgirl, (Case 7 of tree). Apparently well until her seventh year, when her previously normal gait became disordered by adductor spasm, and the spastic gait developed. No sensory symptoms at any time. Knee-jerks excessive; marked ankle-clonus on both sides; no static ataxia. Pupillary reflexes normal; no bowel or bladder difficulty; tendency to talipes equinus.

R. F., male, æt. 7 (Case 8 of tree). Was well, and, as was the case with the others, walked normally until his sixth year, when the same character of spastic gait developed, with the early symptoms of adduction, causing "cross-legged progression." Knee-jerks excessive; slight ankle-clonus. Pupillary, bowel and bladder reflexes normal; no static ataxia. As with the other cases, never any complaint of pain, numbness or anæsthesia.

H. B. F., male, æt. 2 years (son of Case 2, Case 9 of tree). "Never learned to walk right." Has had the spastic tendencies from the earliest efforts to walk, but less marked than at present. He falls easily, and walks with a "spastic waddle." Has difficulty in arising from the floor, but does not "climb up on himself." No muscular atrophy. Child is well nourished; bright mentally; knee-jerks are excessive; no ankle-clonus.

The group of symptoms presented by these cases clearly places the lesion as a primary sclerosis of the crossed pyramidal tracts. The wonderful similarity of the cases, both as to time and mode of onset, absence of sensory phenomena, and (excepting in the first case) state of reflexes, established its hereditary type. It is obvious, therefore, that another congenital affection must be placed in the lists with the already well-known Friedreich's ataxia and pseudo-hypertrophic paralysis. From these two hereditary diseases it differs in the most obvious manner. Nor has it more than a cursory resemblance to the spastic paraplegia of infancy, which is cerebral in origin, involves the upper extremities with the lower, presents cerebral symptoms, and is believed to be absolutely non-hereditary.

It would be of great interest to know the pathological condition in these cases; whether the lesion originates in a vascular defect or is primarily inherent in nerve tissue. This, in the absence of opportunity to examine the cord, must remain conjectural. It is worthy of note, however, that in this family the escape of an individual from the affliction seems to confer immunity upon his or her descendants, the affection travelling in direct and not in collateral lines.

HEMIATROPHIA TOTALIS CRUCIATA. (Deutsche medicinische Wochenschrift, No. 12, 1897.) By M. A. Lunz.

The peculiar affection reported in this paper, dated from the patient's marriage and exposure in field work. It began with periodic pain in the posterior part of the right thigh, which radiated downward into the sole of the foot. After three years, or six years before the report was made, the patient observed wasting of the right lower limb; she had also noticed that the right breast was smaller than the left, and secreted less milk. Three or four years ago the left cheek was observed to be smaller than the right, and at this time the patient began to complain of a pain in the cheek. For a year she had had pain in the back and left lower limb, as well as in the region of the right scapula and right upper limb. At the time of examination by Lunz, the wasting in the skin and muscles of the left cheek was very perceptible. The circumference of the shoulder at the axilla was 43 cm. on the left side, and 41.2 cm. on the right, whereas at the insertion of the deltoid there was no difference on the two sides. On the posterior part of the thorax, atrophy was noticed only at the inner border of the scapulæ. The atrophy on the right side of the abdomen was very perceptible, but still more so in the right half of the pelvis and in the right lower extremity. All movements were well performed, and there was no trace of paresis, but the right leg became tired sooner than the left in walking. The pain was increased by movement. Pressure over the lower lumbar and sacral vertebrae, and over the nerves and muscles of the right lower extremity, caused, at times, sensation of pain. Objective sensation otherwise was not altered. There was no reaction of degeneration. The patellar reflexes, especially the right, were somewhat exaggerated. The process could not have been central, as the hyperæsthesia and atrophy were limited to certain regions innervated by certain nerves. It was evidently a neuritis migrans.

Irritation of sensory nerves acts on the vasomotor centres in such a manner that vascular constriction is produced. Attacks of headache, vertigo, tinnitus aurium, sensation of heat, free perspiration, as well as subjective sensation of cold, and the livid appearance of the lower limbs, observed in this case, were probably due to vasomotor disturbance. The writer believes that the results obtained in noting the surface temperature of the affected and sound parts of the body are in favor of the theory of vasomotor disturbance.

SPILLER.

PHILADELPHIA NEUROLOGICAL SOCIETY.

May 31st, 1897. The President, Dr. Charles W. Burr, in the chair.

Dr. Charles K. Mills presented a

CASE OF FACIAL SPASM (PAINLESS TIC).

The patient, a man fifty-five years old, was brought to Dr. Mills by Dr. Alexander R. Craig, of Columbia. The spasm was of three years' duration, and had gradually increased in severity during this time. All the muscles on the left side of the face, supplied by the seventh nerve, were affected, and at the height of the attacks blepharospasm was very marked. The paroxysms came on at short intervals, and the patient had several during the short time he was before the Society. He had suffered with what he described as "vertigo," which had caused him to fall six or seven times during the last twenty-five years, and he had suffered also at intervals during this time with headache. He had polyuria, but examination showed neither albumin nor sugar. Examination with the ophthalmoscope revealed no changes in the eyeground. For several weeks he had been treated by increasing doses of coniin hydrobromate. One-fiftieth of a grain three times daily was given at first, and the amount was gradually increased until he took as much as the one-fifth of a grain five times daily. The treatment had no perceptible effect in reducing the spasm, and the only constitutional symptoms produced were unusual weakness in walking and a feeling of general debility.

Dr. Wharton Sinkler exhibited a case of

ERB'S PARALYSIS (UPPER ARM TYPE)

in a child of seven years. He stated that the history of traumatism made the diagnosis between Erb's juvenile form of muscular dystrophy and paralysis of the brachial

plexus, of the upper arm type, easier than it would otherwise have been. The patient, a child of seven years, born in a healthy family, attempted two years ago to emulate a trapeze performance which he had seen in the circus. He was found hanging by the arms from a trapeze, with his head down and his legs turned back over the body in a position which produced great strain upon the upper limbs. About two months later the parents noticed that the boy did not use his right arm as freely as the left, and an examination showed that the shoulder and chest muscles of the right side were much wasted, but that no sensory disturbances were present. Pain had not been complained of at any time. The only electrical alterations noted were in the lower third of the pectoralis major muscle, which did not respond to either current. The atrophy was supposed to be due to an injury of the upper roots of the right brachial plexus.

Dr. James Hendrie Lloyd:—This is strikingly like a case that I have in the Philadelphia Hospital. The patient, a man, has complete absence of the pectoralis major and minor muscles. In his case the defect is apparently congenital. I have had occasion to go over the literature, and I have found that these cases are not so extremely rare. My patient is a large, able-bodied man. He has perfect movement of the arm in every direction except adduction across the chest. He has always done hard work and has experienced no disability from the absence of the muscles.

Dr. H. N. Moyer, of Chicago:—I also have seen a case in which the lower portion of the pectoral muscle was entirely absent; while the clavicular portion was intact. I regarded that case as congenital.

Dr. James Hendrie Lloyd presented

A CASE OF TETANY.

The man, an Italian, had only been in this country a few years, and had had a similar attack once before. The symptoms were so characteristic that the diagnosis could hardly be questioned. The case was one of great interest on account of the rarity of the disease in this country.

Dr. Hugh T. Patrick, of Chicago:—In the well-marked cases of tetany which I have seen, the spasm could be produced by the retardation of the circulation caused by simple

elevation of the arm. This would show that pressure on the vessels, as well as on the nerves, may cause the spasm, that is, the continuous cramp which is the usual manifestation of the disease.

Dr. William G. Spiller:—I have seen, I suppose, at least fifteen cases of tetany. This is not an unusual disease in Vienna, where it occurs almost endemically in the early spring months. Pressure on the arm, over the nerves and vessels, sufficiently long continued, usually produces the spasm (Trousseau's sign). This is readily demonstrated in the present case, which is one of the most typical that I have seen. It is a disputed point whether the spasm is caused by pressure on the arteries or on the nerves.

Dr. Dercum presented a case of

UNILATERAL ATAXIA AND TREMOR,

the diagnosis being undetermined, and resting between multiple sclerosis and syphilis.

H. A., aged forty, white, a Swede and a sailor, was a patient at the Philadelphia Hospital.

Family History.—He always enjoyed the best of health until about seven years ago. He has followed the sea for the last twenty-five years. Six or seven years ago he had a chancre, and three years later suddenly lost power in the right side of the body. The hemiplegia was slight and disappeared altogether in four weeks, and was not accompanied by loss of the power of speech. The symptoms of the present trouble, consisting of headache, vertigo, drowsiness and diplopia, first showed themselves last December (1895). About that time he suddenly lost power in the left side of the body, but the attack of weakness was not accompanied by loss of consciousness. He recovered power on this side quite rapidly, but ever since has had difficulty in walking and has not been able to hold objects well with his left hand.

Present Condition.—(Nov. 5th, 1896)—The sway is decidedly increased on making the Romberg test. He stands unsteadily when resting only upon the right foot, and is entirely unable to stand alone upon the left, on account of ataxia and not of weakness. It is observed that the left foot is swung awkwardly upward and outward in walking, and that it strikes with the flat of the sole upon

the ground. The movements of the right foot and leg appear to be normal. The patient, lying upon his back, and being instructed to perform certain movements with his legs, shows marked ataxia of the left leg, and a very slight degree of ataxia of the right. The ataxic movements of the left leg are somewhat jerky in character. Movements of precision are well executed with the right hand, but there is a slight unsteadiness of the right upper limb as a whole. The movements of the left arm are excessively ataxic and jerky. When the patient is told to touch the nose with the tip of the left forefinger, the arm is clumsily moved until it approaches the nose, when lateral, and to and fro, oscillatory movements, jerky in character, supervene, and become more intense with the degree of effort made. Tremor is manifested in the left hand upon voluntary motion, and consists of coarse up and down movements.

The face bears a fixed expression of somnolence; the eyebrows are slightly raised, giving the patient the appearance as though he were making an effort to keep awake. The lines of the face, with the exception of those of the forehead, are comparatively smooth, and the features appear somewhat relaxed, as in paresis. The head is held slightly inclined to the right shoulder. No signs of facial palsy or twitching, and no tremor of the lips are noted. The tongue is protruded slowly and its movements are slightly irregular. The pupils respond normally to tests, and are equal in size. The movements of the eyeballs appear to be as extensive as normal. Marked vertical nystagmus can readily be elicited. The right eyelid seems to droop slightly, and the right palpebral fissure is smaller than the left. Neither anesthesia nor affection of the sphincters is noted.

Speech is somewhat scanning and drawing and reminds one of that of multiple sclerosis. The knee jerk is plus on the right side and normal on the left. There is slight ankle clonus on the right side, but none on the left. The elbow jerk, biceps jerk and wrist jerk on the right side are plus; on the left side they are normal.

The plantar reflexes are marked, the left excessively so. There is no toe reflex. The superficial reflexes of the thigh and abdomen are much increased. The patient had headache, dizziness and ringing in the ears for five years, until the fall of 1895, when he was relieved by medicine, and has

no dizziness now. He has no attacks of any kind, and no pain in the bones. His mental condition is fair. He can give a good account of his past life and of his present symptoms; he is neither expansive nor depressed at any time, has no headache, and has never had any delusion.

Dr. Dercum thought that the case looked at first sight very much like one of multiple sclerosis with unilateral symptoms. In favor of this view were the coarse intention tremor and the scanning speech. If this view is correct, it would, perhaps, point to a possible relation between multiple sclerosis and syphilis. The syndrome, however, is certainly not that of syphilis, and whatever the character of the lesion may be, it is not in all probability that of ordinary nervous syphilis. The question of paresis was also taken up and briefly considered, but this diagnosis was considered improbable.

Dr. Wharton Sinkler:—This case recalls a patient who was under my care some years ago, and suffered from disseminated sclerosis with marked intention tremor on both sides. He had a cerebral hemorrhage with hemiplegia on the left side, which developed while he was under treatment in the Philadelphia Hospital for the multiple sclerosis. He partially recovered from the paralysis and the tremor disappeared entirely on the hemiplegic side, while it remained unchanged on the other. May there not be a distribution of the sclerotic patches in areas which inhibit the intention tremor on the one side in Dr. Dercum's case?

Dr. Henry S. Upson, of Cleveland:—It is a well-known fact that lesions in the great basal ganglia may cause intention tremor. Lesions about the optic thalami may cause symptoms similar to those of paralysis agitans. This would suggest that, perhaps, in this case there is a lesion near or in the great basal ganglia which causes these symptoms. It may be impossible to make a diagnosis between such a lesion as I have spoken of and the sclerotic patches of ordinary insular sclerosis.

Dr. F. X. Dercum presented a case of

RIGHT HOMONYMOUS HEMIANOPSIA

without visual or optic aphasia, following an apoplectic attack. The history of this case is briefly as follows:

H. H., a patient at the Jefferson Hospital, aged forty-five, was seized with dizziness and vomiting two years ago while at work in his garden. He soon became unconscious

and remained so from ten o'clock in the morning until three in the afternoon. On recovering consciousness he found himself in bed. He was able to speak, but had some weakness of the right side. A curious optic illusion was, however, present; he felt as though the ceiling were much lower, and were arched over his bed. This illusion persisted for several days. A subjective numbness of the left leg below the knee was also noticed.

At present the patient presents a typical right homonymous hemianopsia with preservation of central vision, but no hemiplegia, and no anesthesia. He states that the sense of numbness, which made its appearance in the left leg in the beginning of the attack, has steadily persisted. It does not appear to be pronounced, and cannot, of course, be verified by any physical examination. The heart and vessels are normal. The case is interesting as an instance of an apoplexy, in all probability resulting from a cerebral hemorrhage, in which every symptom, save the hemianopsia, has disappeared; and also because of the absence of visual or optic aphasia.

Dr. Quimby, of New Jersey:—It seems to me that the sudden attack would point very strongly to a diagnosis of hemorrhage. I have seen a similar case in which the necropsy showed a cortical hemorrhage.

Dr. Charles W. Burr presented

A CASE OF HEMIPLEGIA (POSSIBLY HYSTERIC) WITH
ANKLE CLONUS.

The patient was a single man about twenty-seven years old and a laborer. He denied having had syphilis and having indulged in alcoholic excess. In September, 1892, while at work he suddenly became dizzy, fell down unconscious and remained in this condition for six weeks. On return of consciousness he was found to be speechless and paralyzed on the left side. His jaws were so stiff that he had to be fed with a spoon. After three months speech returned, and at the end of a year he could walk. Since the beginning of his illness he has had several attacks of unconsciousness. At the time of his admission to the Philadelphia Hospital, in 1895, he appeared to be a case of ordinary hemiplegia, and was sent to the out-wards. Some

months later he had an attack in which he screamed, threw himself on the ground, writhed, jerked and bit, but did not become unconscious. He was sent to Dr. Burr's ward and became quiet in a few minutes after his arrival. For some days he lay apparently perfectly conscious and seeming to hear and understand all that was said to him, but unable or unwilling to move or speak. On the eighth day he became very religious in his actions, clasped his hands and looked heavenward or held an open prayer-book hour after hour without turning the leaves. The next day he was sentimental and wished to kiss the hands of those about him, and finally was somewhat maudlin. Speech then returned, and at first he spoke in whispers, but in a few hours he talked well and got up and walked about the ward as usual.

Examination showed that he was a strong, well-built man. The heart, kidneys and, indeed, all the thoracic and abdominal organs, were normal. Very marked spastic hemiplegia of the left side was noted. The left knee-jerk was exaggerated. There were regular and persistent wrist, patellar and ankle clonus, but no muscular wasting. The sensory symptoms were remarkable; at times there was complete left hemianesthesia, stopping abruptly at the median line and involving the mucous membranes; at times this anesthesia disappeared completely, and at other times affected only a part of the leg or arm, the distribution then having no regard to nerve supply, but resembling a glove or stocking. The same was true of the pain sense, which, however, was never completely absent. At first the eye examination was negative, but later Dr. Oliver discovered contraction of the fields, involving especially the left side, more marked in the left eye, and affecting chiefly the left upper quadrant. The paralytic symptoms have remained constant, but, as stated, the sensory have varied greatly.

Dr. Burr thought the man presented many of the symptoms of hemiplegia, but that this diagnosis did not explain all his symptoms. The fit certainly was hysteroidal, and he knew of no organic disease which would account for the strange variation in the anesthesia. He believed, therefore, that the man had hysteria, but was by no means sure that it was not added to an old organic hemiplegia. Until recently he would have said that the great increase in the deep reflexes, and especially the existence

of true ankle clonus, were absolute proof of the organic origin of the hemiplegia, but he was beginning to be more than doubtful of the truth of that view, and had presented this man to obtain an opinion on the question.

Dr. Charles K. Mills:—I do not believe that this is a case of hysteria pure and simple. I believe that the man is suffering from an organic disease with possibly hysteria superadded. I would say in regard to his vacillating symptoms, that I have seen variations in the sensory phenomena in cases of syringomyelia similar to those presented by this man.

Dr. Hughes, of St. Louis:—Some years ago I was interested in a medico-legal case in which Dr. Moyer was also concerned. In this case, the question of ankle clonus was an important element in the diagnosis. I have taken the position that the profession has been largely at fault on this matter of hysteria. The general ruling in diagnosis is either all hysteria or no hysteria, yet it is only reasonable to expect, and if we observe our cases closely we will find it true, that the gravest kind of lesions bring out latent hysterical manifestations. I think that we have done ourselves and our patients great injustice in assuming that hysteria may not coexist with grave organic disease. Gowers has taken the same position. I believe that if in this case the history is carefully inquired into there will be found a hysterical tendency in the family. I do not doubt the existence of organic lesion in this case.

Dr. H. N. Moyer, of Chicago:—The case referred to by Dr. Hughes was a very interesting one. The question was whether the trouble was functional or organic. I leaned to the view that it was organic. The patient had ankle clonus. The man's present condition after a period of ten years confirms the diagnosis of organic lesion. He is in the condition of a person after a moderately severe attack of transverse myelitis.

Dr. Hugh T. Patrick, of Chicago:—The question of clonus interests me greatly. I have examined a number of cases of hysterical clonus. The clonus in this case seems to be that of hysteria. It started at the rate of about three hundred and sixty movements a minute, which is the ordinary rate of organic clonus, but before the termination of a third of a minute it had fallen to a hundred and twenty a minute. As far as my experience goes this change in the rate is characteristic of hysterical clonus.

Dr. J. H. W. Rhein:—Apropos of the question of ankle clonus in hysteria, I should like to present again the case reported by me at the last meeting of this Society. The history is briefly as follows: The patient, after inhaling benzine vapor while cleaning a manure tank, became partially unconscious, and soon afterwards had tremor in the entire body. A month

later, when first seen by me, he had spasticity in the legs, general tremor when walking, spasmodic strabismus, segmental anesthesia, and a true ankle clonus, which persisted on one side, but was easily exhausted on the other. Under hypnotism the ankle clonus disappeared. Dr. McCarthy, who has hypnotized the patient frequently, saw it disappear under suggestion. It is not now present. As the patient walks you will notice that he has lost the spastic gait and the general tremor. All the symptoms, including the segmental anesthesia and the spasmodic strabismus, have disappeared. The diagnosis is clearly hysteria and the case demonstrates beyond a doubt that in this affection we may have a true ankle clonus.

Dr. A. A. Eshner:—I should like to ask a question which suggests its own answer. If hysteria may develop entirely apart from organic disease, is it less likely that it should occur in connection with organic disease, and need the presence of organic disease exclude the existence of hysteria, or the presence of hysteria exclude the existence of organic disease? My view of Dr. Burr's case is that it is organic, whether primarily or secondarily, I cannot say. Whether, in addition, there is hysteria, it is difficult to determine, but I am inclined to think that there is.

Dr. Martin W. Barr, by invitation, exhibited

AN EXTRAORDINARY CASE OF ECHOLALIA.

Dr. Charles K. Mills:—Had we time this paper would open up a wide field for discussion. It seems to me that Dr. Barr, like myself, has perhaps described two affections which are radically different, although I do not know that he intended to include the case exhibited in the first class referred to by him. The cases of echolalia and of ejaculatory speech of various kinds, where the attacks come in paroxysms, are more of the nature of obsessions, and are not cases of true echolalia, as this seems to be.

One other point. Probably no one has taken more interest than I have in the attempt to localize special parts of the brain related to the function of speech, yet in a case of this kind too much stress may be laid upon the effort to definitely fix the position of the lesion to explain the symptoms. The case would seem to me to be better explained by the supposition of arrest of development of large areas of the brain, although, of course, even in this condition, there may be a preponderance of the arrest, or of the lesion, in certain parts.

Dr. Charles K. Mills exhibited

A CASE OF PARALYSIS OF THE ELEVATORS OF THE EYEBALLS.

J. F. B., aged thirty-seven, white, single, and residing in West Chester, Pa., was sent by Dr. Ellwood Patrick to Dr. Mills for consultation.

The patient's ocular disturbance began four years ago, and vision had been getting weak. He had had diplopia during the past year, one image being situated a little above the other. There had been no history of pain. Since last fall there had been disturbance of locomotion, and the patient had had difficulty in raising his feet from the ground. Visual symptoms had been noted for two years. The man could not raise his eyeballs above the horizontal plane in attempting upward rotation, and in doing this one or the other eyeball tended to move slightly outward. Lateral and downward movements appeared to be good. The movements of the upper eyelids seemed to be well preserved, but the inability to move the balls upward gave the appearance of partial ptosis. The patient complained of a general feeling of weakness in the legs. He had not had lancinating pains, and his knee-jerks were exaggerated, and although there was no persistent ankle clonus, a slight spurious one was noted on the left side.

Dr. Hansell reported that there was decided concentric limitation of the right field, and positive, but slight, concentric limitation of the left. The paralysis of the upward rotators was complete. There was divergence of the left eyeball, but no lateral paralysis. The pupils were equal and reacted sluggishly to light and in accommodation individually and consensually. The media and fundus in each eye were healthy. The central visual acuity was 20/40 R. 20/30 L. Diplopia, with the second image a little above the first, and to one side, was noted when the objects were in certain positions. The gait was peculiar, in that the patient did not follow a straight line, which was possibly due to false projection. There was slight, almost imperceptible, difficulty in speaking. The tongue was protruded somewhat to the left, and there was some tendency to drooling.

Dr. Joseph Sailer reported

A CASE OF GLIOSIS CEREBRI.

Dr. Wm. G. Spiller read a communication in the name of Dr. G. Marinesco, of Paris, on

THE PATHOLOGY OF MORVAN'S DISEASE.

The case of Morvan's disease, which I have reported to the Société des Hôpitaux, was a well-defined form of the disease, and affords, in my opinion, a solution of the pathology of this affection. The clinical diagnosis was made by Charcot. The pathological examination, made by me, has only revealed a lesion of the gray matter, involving especially the posterior horn, and, to a slight degree, the anterior also, at least in the lower cervical region. In the portion of the cord affected in this case is the centre for the sensory, motor and vasomotor innervation of the hand. The nerves are intact. I have been led to conclude, therefore, that the trophic disturbances of Morvan's disease in my case should be referred to the most important lesion, viz.: the alteration of the posterior horn. Furthermore, in this case the lesion was unilateral and on the same side as the trophic disturbances, the nerves were intact, and the lepra bacillus was not found. How can these conditions be otherwise interpreted? A question might only arise as regards the alteration of the anterior horn. The condition here may possibly have had some effect on the symptoms, but the changes in this horn were not very important, and their location does not explain the trophic disturbances. One is forced, therefore, to admit that the cause of these trophic lesions is to be found in the condition of the posterior horn or, more exactly, of the intermediate and posterior gray matter. In the light of our present knowledge it seems to be an established fact that the alteration of these regions may cause cutaneous and osseous trophic disturbances. These, however, are produced reflexly, as I have shown several times in my papers. Leyden and Goldscheider, and Brissaud have adopted my views.

Although I have determined that Morvan's disease is due to destruction of the posterior gray matter, it does not

follow that other lesions cannot cause an analogous clinical picture. The explanation which I offer in regard to the nature of Morvan's disease in my case seems to me the only possible one. At all events, the symptoms cannot be attributed to an abnormal condition of the peripheral nerves, as these were intact.

Dr. Spiller:—Dr. Dercum and I have been unable to believe that in every case in which the posterior horn is destroyed in the lower cervical and upper thoracic regions the symptoms of Morvan's disease are present. We reported, in the *American Journal of the Medical Sciences* (December, 1896) a case of syringomyelia in which the posterior horn of one side was destroyed throughout the cervical region, and also in the upper thoracic region, but no symptoms of Morvan's disease had at any time been detected. Dr. Marinesco has made so many valuable contributions to neurological literature that we could not lightly pass over an apparent discrepancy in our findings and the interpretation of them. We have, therefore, requested an explanation and he has kindly sent the above statement.

We cannot judge from our case of the nature of the lesions produced by destruction of the intermediate gray matter, that is, of the substance between the anterior and posterior horns, as the cavity does not extend far into this region. It is true, however, that most of the fibres passing into this portion of the cord from the posterior roots are destroyed, but if trophic cells are situated here their axis cylinders may pass to the periphery of the body by way of the anterior roots. This may possibly explain the differences in Dr. Marinesco's case and ours.

In another communication Dr. Marinesco has called our attention to the fact that in our case an arthropathy of the shoulder existed on the same side as the cavity in the posterior horn, and, if we understand his meaning correctly, he attributes the disease of the shoulder joint to the destruction of the posterior horn. This may possibly be the correct interpretation of our case, but we prefer to be somewhat cautious in the full acceptance of this explanation until other cases are reported.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. HENRY L. SHIVELY, M.D., N. Y.
WM. C. KRAUSS, M.D., Buffalo, N.Y. A. STERNE, M.D., Indianapolis.
W. M. LESZYNSKY, M.D., New York.

NEUROPATHOLOGY.

ASCENDING NEURITIS FROM SCAR PRESSURE.

M. M. Carrière et Rainguet (*Gaz. Hebdomadaire de Médecine et de Chirurgie*, May 27th, 1897) reported a case of ascending neuritis following traumatism.

E. L., aged forty-six, farmer, entered the hospital a few days ago for pain in the left foot and ulceration of the little toe.

At ten years of age he gave himself a blow with a hatchet at the top of the fourth left interosseous space, about 5 c.m. in length. A month after, the wound had healed.

In the course of the third year, the patient began to suffer from tumefaction of the great toe. Some days afterwards the pains appeared at the top of the little toe. From this time the attacks of pain occurred from time to time, and in the month of March, the patient having ascertained that the condition was one of progressive necrosis of the left little toe, decided to enter the hospital.

After having eliminated, by a careful examination, tabes, Raynaud's disease and Friedreich's disease, the case was decided to be one of ascending neuritis of traumatic origin, of which the causal agent was the blow already spoken of.

The point commended upon was the tardy appearance of the results; and the fact is explicable only in admitting the production of a hardened cicatrix, the contracted tissue of which had surrounded the nerve supplying the little toe, thus producing an ascending neuritis.

MITCHELL.

PATHOLOGIE GÉNÉRALE DE LA CELLULE NERVEUSE, LÉSIONS SECONDAIRES ET PRIMITIVES. Marinesco. (*La Presse Médicale*, April 27th, 1897.)

Referring to a previous article, in which he set out to show that lesions of the nerve cells secondary to nerve section have a special character which distinguishes them from those due to causes acting primarily upon the cell itself, Marinesco returns to the same task, supporting his conclusions upon the results of newer experiments by himself and others. Section of a nerve is followed by modification of the cells of its centre of origin, which constitutes what he calls "reaction at distance." This reaction occurs in all animals—

though its rapidity depends upon the sort of animal—and takes place always in a uniform manner. Three phases are to be distinguished: that of reaction, that of degeneration, and—where the cell recovers—that of repair. In the structure of the cell he recognizes three elements: (a) the fundamental achromatic substance, (b) corpuscles having affinity for basic dyes, the chromatophile elements, and (c) a fibrillary or striated substance. The alterations affect especially the achromatic substance—"tropho-plasma," and the chromatophile elements—"kynéto-plasma." The phase of reaction begins with disintegration of the chromatophile elements, especially about the point of origin of the axis cylinder. In a later stage, this "chromatolysis" extends to the greater part of the cell, and the nucleus is found to have moved from the centre of the cell to its periphery. As the change proceeds, the delicate striation of the cell and of its protoplasmic processes is lost, and the chromatophile elements are reduced to a fine dust. Section of a sensory nerve is followed by similar changes in the cells of its ganglion.

When the sensory root is cut peripherally from the ganglion, the changes are much greater than when the portion between the ganglion and cord, or the posterior columns of the cord are cut. Certain cells being unable to recover, the change in them goes on to degeneration, and they atrophy and disappear. To determine how repair takes place, Marinesco examined the medullæ of rabbits, killed 24, 46, 63 and 90 days after section of the hypoglossal nerve. The phenomena of repair already marked at the end of 24 days, consisted in hypertrophy of the affected cells, with new formation of the chromatophile elements, beginning especially about the nucleus which still occupied an eccentric position. By their great size, and their deeper staining, the regenerating cells could be distinguished even under low magnifying power, from those of the unaffected nucleus. This slow hypertrophy was shown through the whole series of observations, and was still going on 90 days after the nerve section. The author thinks that the same changes in the cells occur as a result of neuritis. Selecting the large cells of the anterior horn of the cord for sake of uniformity, he has studied their primary changes in experimental anæmia, hydrophobia, botulism and tetanus. The lesion in each case he describes somewhat at length. Summarized, they affect both chromatophile and achromatic substance. "Chromatolysis" may be peripheral, perinuclear, or diffuse, the different forms predominating in different diseases. The nucleus has not the same tendency to seek the periphery of the cell as in secondary lesions. The changes in the achromatic substance are two-fold—molecular disintegration, and coagulation and transformation into a glassy material. Another modification is increased affinity for dyes on the part of the achromatic substance. A change to which the author attaches much importance, is an increase in number of the neuroglia cells, which seems to be in direct relation to the amount of change in the achromatic substance. Alteration of the neuroglia and the vessels he finds pretty constantly present in primary lesions, while in lesions secondary to nerve injury they are absent. On this latter fact, and on the difference in character in cell alterations, he feels able to distinguish the two classes of lesions, primary and secondary. The different appearances of the cells in these two classes of lesions, and also in different diseases, are illustrated by enlarged colored figures, showing the cell as it appears when stained by Nissl's method. In conclusion, the author emphasizes the importance of the pathology of the nerve cell, and expresses the hope that its further study may lead to the establishment, some day, of a system of cellular therapeutics.

C. L. ALLEN.

CONCERNING INJURIES FROM ACCIDENTS AND MUSCULAR ATROPHY.
With Remarks on the Laws relating to Legal Recompense after
Accidents. (Berliner klinische Wochenschrift, No. 12, 1897.)
By F. Jolly.

The patient's left arm was torn away near the head of the humerus, but the wound healed without difficulty. The man was afterward employed in cleaning machinery, and was obliged to lift heavy weights with his right arm. About two years after the accident he was forced on account of atrophy of the muscles of the right shoulder, to seek other employment. The deltoid was much wasted, and abduction and elevation of the arm were impossible, and reaction of degeneration was also noticed in this muscle. The right supraspinatus muscle was affected. Fibrillary contractions were observed in the deltoid, trapezius, biceps and triceps. The deformities, which existed in the lower extremities were attributed to anterior poliomyelitis, and to an accident which occurred in childhood. It is very probable that the atrophy in the right shoulder was due, at least indirectly, to the loss of the left arm, and possibly to overwork. It is not improbable that the muscles of the right arm were predisposed to degenerative atrophy on account of the anterior poliomyelitis, although no alterations had been observed in this limb before the accident. In the atrophic area and somewhat beyond this, sensation was much affected, especially for pain and temperature. It is possible that the tearing away of the arm may have caused small hemorrhages in the cord, though as the patient had had hysterical convulsions, the anæsthesia may have been hysterical. The balance of the paper is devoted to the subject of legal recompense after accidents.

SPILLER.

ABOLITION OF THE REFLEX OF THE TENDON OF ACHILLES IN SCIATICA.

J. Babinsky (Gazette des Hôpitaux, No. 100, 1896) shows that in healthy individuals the tendon reflex of the Achilles tendon is normal, while in disturbances of the sciatic nerve the reflex is either abolished or greatly diminished. He found this phenomenon not only in cases of intense sciatica with marked muscular atrophy (sciatic neuritis), but also in the lighter forms of ischialgia, designated sciatic neuralgia.

The author refers to two cases where the difference between the behavior of the Achilles tendon reflex on the sound side and the affected side was very conspicuous, being totally abolished on the affected side, while on the unaffected side it was normal. This sign, the author concludes, is a valuable diagnostic aid; it indicates the existence of some organic change in the nerve, and excludes the hypothesis of simulation, also aids in differentiating between a true and an hysterical sciatica. In the latter, according to Babinsky, the symptom would be wanting.

KRAUSS.

PROGRESSIVE, MULTIPLE, LOCALIZED NEURITIS (Mononeuritis Multiplex). (Deutsche medicinische Wochenschrift. No. 5, 1897.)
By E. Remak.

Prof. Remak reports the case of a type-setter with amyotrophic paresis and reaction of degeneration in the muscles innervated by the left ulnar nerve. The man had never suffered from lead poisoning. Improvement was noticed after the use of the galvanic current. The patient began to have difficulty in raising his right thigh, and paræsthesia in this part three months after the beginning of the paralysis in the upper limb. A localized, peripheral neuritis developed in the motor nerves of the iliopsoas muscle and in the anterior crural and obturator nerves. These nerves come from the third and fourth roots of the lumbar plexus. The process was shown to be a peripheral one

by the involvement of the sartorius muscle, which is not paralyzed in poliomyelitic paralysis, and of the sensory fibres of the anterior crural nerve, and by the normal condition of the tibialis anticus. Still later the ulnar nerve of the right hand was affected. The course of the disease justified the diagnosis of progressive polyneuritis, probably idiopathic in origin. Lead was not thought to be the cause of the trouble, because the paralysis progressed after the man had stopped working with type, and because saturnine paralysis, when in rare cases it appears in the lower extremities, never attacks the thigh muscles alone. Lead paralysis also is very uncommon in type-setters. The case is peculiar in its course and want of symmetry, and the name of mononeuritis multiplex may well be applied to it. As the ulnar was the only nerve affected for three months, the impropriety of entirely separating localized neuritis (mononeuritis) from the multiple form is well demonstrated.

SPILLER.

A CASE OF SO-CALLED PARAMYOCLONUS WITH PHENOMENA OF COM-PULSION. (Berliner klinische Wochenschrift, No. 44, 1896.) By L. Stembo, M.D.

Stembo reports a case in which all the cardinal symptoms of myoclonia, as given by Friedreich and Unverricht, were present. Coprolalia as seen in the *maladie des tics convulsifs*, was also observed. There was no echolalia. The writer can only refer to one similar case. This was reported by Spitzka.

Dr. Stembo regards his case as a proof of the impossibility of making a sharp distinction between the various forms of convulsive movements. These occur in many different nervous diseases, and especially in hysteria. Myoclonia in most cases is of a hysterical nature, even though no other stigmata may be present. Monosymptomatic hysteria is by no means uncommon. Some of these convulsive movements may be due to organic disease, but in no case are they more than symptomatic. The writer warns us against the too strict separation of the different forms of spasmodic movements.

SPILLER.

HEMIPLEGIA AND EPILEPSY.

M. Féré (La Méd. Moderne, May 5th, 1897) briefly reported to the Société de Biologie a case of hemiplegia and epilepsy. His observations were upon a case of epilepsy in which there had been a cerebral hemorrhage, resulting in left hemiplegia. In the attacks of epilepsy which followed it was remarked that the hemiplegic side remained unaffected by the convulsions which continued to manifest themselves as before upon the right side.

MITCHELL.

PSYCHOLOGY.

EXPERIMENTS ON MEMORY TYPES. Psychological Review, May, 1897.

Chauncey J. Hawkins (Yale) tested auditory and visual senses in public school and college students. For the auditory tests he read aloud three groups of three numbers each at the rate of one number per second. The first group was read once, the second was read twice, and the third group three times. He found that the second reading nearly always weakened the memory a trifle, that the subjects could not recite the reading quite so well as they did after the first reading. The third reading produced a very marked improvement. He also observed that the younger pupils nearly always reproduced the numbers without hesitation, while the college students always hesitated and required more time to reproduce the list.

In his visual tests two lists of words were placed upon the black-board and covered. The first test was then exposed for thirty seconds, and the second list was exposed at the rate of one word every two seconds (thirteen short familiar words in a list). This test was tried on pupils ranging from eight to fifteen years of age, and revealed the fact that successive visual memory is much better in the younger pupils than simultaneous visual memory, there being a difference of 22 per cent. at the age of eight, while there was a difference of only 1 per cent. at the age of fifteen. In students from fifteen to twenty years of age it was found that simultaneous exposure gave a much better memory than successive exposure.

Auditory memory was found to be much better than visual memory in pupils from eight to twelve years of age, a fact probably due to their being accustomed to a large amount of auditory work. A like reason, probably, accounts for the fact that students ranging from fifteen to twenty years of age showed a much better visual than auditory memory.

CHRISTISON.

"REACTION TYPES." *Psychological Review*, May, 1897.

Farrand (Columbia) tested the two noted pianists, Rosenthal and Sieveking, as to their reaction time to sound. The Hipp chronoscope was used, and they were to raise the two forefingers of the right hand when the signal was given by a tap with a metal hammer. Neither subject knew that the other was to be tested. Rosenthal made seven records as follows (times in 0): 110, 118, 119, 112, 119, 123, 123, giving an average reaction time of 117.7, and an average variation of 3.8. Sieveking's reaction time was 114, 114, 117, 120, 108, an average of 116.6, and an average variation of 2. No instruction as to attention was given either subject, but Rosenthal declared his entire attention was on the signal, while Sieveking was as positive his attention was on his reacting hand. Rosenthal repeated the experiment, this time giving his attention to his reacting fingers instead of the signal. The result was, 250, 230, 270, 268, an average of 254.5, and an average variation of 14.5. Sieveking was asked to react with his attention on the signal instead of on his hand as before. He attempted to do so, but declared it impossible and declined to proceed. The musical training and characteristics of these two artists are said to be diametrically opposed.

Cattell (Columbia) thinks these experiments support the idea "that people react most quickly and regularly in the way in which they were used to reacting, and that when the reflex character of the reaction is disturbed, the times become longer and more irregular."

Baldwin (Princeton) remarks, in opposition to Wundt: "The existence of 'types' of simple reaction can no longer be ignored by anyone."

CHRISTISON.

IS TRIONAL A USEFUL HYPNOTIC, AND DOES IT POSSESS ANY ADVANTAGES OVER SULFONAL? By Prof. J. Von Mering, Halle (New Eng. Med. Monthly, Jan., 1897).

It has been observed that in certain individuals sulfonal has a cumulative action leading to a severe disturbance of the general health (hæmatoporphyria), which in some cases lead to a fatal issue. In many cases in which the dosage had been carefully regulated the action was continued beyond the desired period. Experiments showed that trional has all the useful properties of sulfonal in a higher degree, while the undesirable effects are absent or less marked. Morro found that trional was more easily and completely decomposed within the organism, and that the ultimate products were more easily eliminated. Since the introduction of trional six cases of toxic phenomena have been observed. In two or three of these hæmatoporphyria was found in

the urine. Beyer concludes that the toxic phenomena are not wholly due to the specific action of the trional, but must in part be referred to other causes and complications. No fatal case from a single large dose has yet been reported, although such must be possible, judging from experiments on animals. Three observers noted excretion of hæmatoporphyrin in human subjects who had never taken either sulfonal or trional. In one case of severe anæmia this was observed. Therefore it is questionable whether this is to be considered as a direct result of the action of these agents. From personal experience and that of sixteen distinguished colleagues, it follows that we have no hypnotic to be preferred to trional. In only one of the reports received has it been ranked as on equality with sulfonal. Some have ceased to use sulphonal. It is true that hæmatoporphyrinuria has been observed in a few cases after trional also, but it is much more rare and is easily avoided and disappears when the drug is stopped.

In the sleeplessness following bodily pain trional may also give good service when 1.0 gm. is combined with 0.005 gm. of morphine. In spite of all investigations, we have not arrived at any explanation of the occurrence of hæmatoporphyrinuria. It may be regarded as proven that in the human subject it is more easily produced by the long-continued use of sulfonal than trional, and that when it does appear other factors must always co-operate, such as deficient nutrition, pre-existing depreciation of the general system, or some other unknown factors. Of the hypnotic and sedative drugs now at hand it can be safely said that for the greater number of cases for which they are appropriate, trional will be awarded first place. The complications and other effects of trional usually are lighter in degree, and by the exercise of a little caution can be avoided as far as any practical importance is concerned. A continuous protracted daily administration of trional should be abandoned: it is almost always superfluous. If a continuous sedative is needed, the administration of trional should be varied by the use of other hypnotics. When there is a protracted effect and somnolence on the following day, it is an indication to lessen the dose. In the great majority of cases 1.0 gm. doses are quite sufficient. If it is necessary to increase the amount, there is often a brilliant result by raising it merely 0.25 gm. There are some cases requiring 2.0 gm., but these are very rare. That a temporary suspension of the medication is much more necessary in the case of large doses than with smaller ones is self-evident.

FREEMAN.

SECONDARY DEGENERATIONS FOLLOWING FOCAL HEMORRHAGES.

Drs. G. Dotto and E. Pusateri, of Palermo, have undertaken to study the alterations in the nerve cells of the cerebral cortex secondary to intracerebral focal hemorrhages, and the connection between the cortex of the island of Reil and the capsula externa in man. (*Rivista di Patologia Nervosa e Mentale*, Jan., 1897). As a result of their studies they feel warranted in drawing the following conclusions:

1. That following intracerebral focal hemorrhages there are secondary atrophic processes in the cerebral cortex of the hemisphere of the same side.
2. That these alterations are not uniformly diffuse, and interest in different degrees the nerve elements.
3. That the cortex of the island of Reil in man is in connection with the external capsule.

Book Reviews.

DIE PATHOLOGIE DER TABISCHEN HINTERSTRANGSERKRANKUNG (The Pathology of the Tabetic Disease of the Posterior Columns). From the laboratory of Prof. Obersteiner, in Vienna. By Dr. Emil Redlich, Docent in the University of Vienna. Gustav Fischer, Jena, 1897.

Any one ignorant of the extent of our knowledge on the pathology of tabes dorsalis, would be surprised by the size of the volume which Redlich has written. No author is more capable than he to treat this subject in an interesting manner, and the book will be prized by every one concerned with the pathology of the nervous system.

The first part is devoted to the normal anatomy of the posterior roots and posterior columns. Redlich has not been able to observe the bifurcation of posterior root fibres in adult man, though he does not deny its existence. The descending degeneration, observed in the posterior columns in some cases in which the posterior roots are diseased, may be in the descending fibres of the posterior roots and not, necessarily, in the descending branches of posterior root fibres. Redlich doubts the existence of posterior root fibres in the posterior commissure of the cord, or rather, he believes that if such fibres are present here their number must be limited. He cannot find posterior root fibres in the anterior commissure, nor in the opposite posterior, nor in the opposite anterolateral column. Naturally, he is not speaking of neurones of another order.

The fibres in the posterior columns which degenerate downward (comma zones and oval field) may be exogenic or endogenic, i. e., from cells without or from those within the cord, but it is more probable that they are posterior root fibres.

Redlich has seen the degeneration of the ventral fields in cases of lesions of the cauda equina, as described by the reviewer in collaboration with Prof. Dejerine. It is difficult for us to understand how any one could state that the ventral fields in the thoracic cord contain only endogenic fibres. Redlich acknowledges that in advanced cases even of tabes these fields are not intact, though he has never found them intensely degenerated. The statement made by him that the dorso-medial bundle (oval field of Flechsig) in the lower sacral cord unites with the ventral field, is one which the reviewer can fully confirm. While working in the laboratory of Prof. Dejerine, in 1895, he had the opportunity to examine the sacral region in a number of tabetic cords and observed the condition to which Redlich refers. The latter believes that this union does not point to identity of fibres in the two zones.

It is interesting to know that the fibres of the columns of Burdach in the cervical region of an eight months' fetus are more medullated than those in the columns of Goll, but Redlich does not say that this has any physiological significance.

The second part of the book is devoted more exclusively to the changes observed in tabes. The writer discusses the question as to whether the degeneration of the posterior columns is systemic, in the sense of Strümpell and Flechsig, or an elective process in the fibres of the posterior roots (Mayer), or a segmentary form of posterior root degeneration involving equally all the fibres of the affected roots (Leyden, Marie, Dejerine, Redlich).

An interesting case of tabes is mentioned by Redlich. The Achilles tendon reflex was absent and vesical disturbance and preservation of the patellar reflex were noted. The "local tabetic degenera-

tion" (in the zones occupied by the posterior roots immediately on their entrance into the cord) was limited to the sacral and lower half of the lumbar cord.

In the cases of tabes in which the dorsomedial bundle was found degenerated Redlich observed "local tabetic degeneration" in the posterior columns above the degenerated dorsomedial bundle, and he believes that posterior root fibres are present in this bundle. In this sense, perhaps, it is allowable to speak of the "posterior median root zone."

The explanation which Redlich gives for the cases in which a moderate degeneration in the lower part of the cord causes little or no ascending degeneration is probably correct. Where degeneration in the lower part of the cord is slight it disappears as the collaterals of the posterior root fibres are distributed to the cinerea.

The cases of tabes in which isolated, band-like areas of hypesthesia are present are those in which the "local tabetic degeneration" occurs in certain roots in the middle and upper thoracic cord. It has seemed to the reviewer, before reading Redlich's book, that this is the most probable explanation for these areas which have become so prominent in cases of tabes and syringomyelia.

The pain sense is usually affected in tabes before the tactile. There are no fibres exclusively for the conduction of pain (Goldscheider, Redlich). Pain is the result of the summation of irritation, and as certain of the fibres from a given area may be destroyed in tabes while others persist, this irritation may be lessened in intensity and be perceived as tactile sensation, but not as painful.

The possibility of the development of tabes from infectious diseases, chronic poisoning, as from lead, and taking cold, cannot be denied. Trauma in a very few cases may have some etiological relation to tabes. Overexertion, especially of the lower limbs, may be of importance in the development of the disease in question. The writer does not believe that tabes may begin in ascending peripheral neuritis, but he thinks that the disease of the nerves is caused by the same agent as that of the posterior columns, and one process is not secondary to the other.

Redlich has found changes in the cells of the spinal ganglia, but they are not sufficient to explain the alteration of the posterior roots and are not constant. The writer, therefore, maintains the correctness of the view advanced by Obersteiner and himself (degeneration of the intramedullary portion of the posterior roots).

The literature quoted is extensive, and the illustrations are numerous. SPILLER.

THE STATEMENT OF STELLA MABERLY. A Novel. F. Anstey. D. Appleton & Co., New York.

Mr. Anstey has turned the point of his pen, which has hitherto good-humoredly pricked the foibles of his fellow Londoners, to a serious undertaking. He has made it a mental lancet to metaphorically lay bare the workings of a diseased mind, and he has done it with such skill, that where the average reader sees only a simple tale of English rural life, with a horrible tragedy for its climax, the physician sees a perfect picture of a paranoiac. The novelist who attempts to portray character made abnormal by disease is too apt to draw on his imagination for literary effect; but in the delineation of this character, with its hereditary flaw and its growing delusions and hallucinations, Mr. Anstey has worked out a careful and accurate study of characteristic methods of reasoning, that lead a paranoiac to a violent deed.

JELLIFFE.

TELEPATHY AND THE SUBLIMINAL SELF: An Account of Recent Investigations Regarding Hypnotism, Automatism, Dreams, Phantasms, and Related Phenomena. By R. Osgood Mason, A.M., M.D., Fellow of the New York Academy of Medicine. Henry Holt & Co., New York, 1897.

Dr. Mason is known as a very sincere and earnest student of the subject with which this book is concerned, and a systematic work from his pen naturally arouses much interest. The author deals first with the subject of telepathy or thought-transference; then with hypnotism, clairvoyance, double personality, somnambulism, and the various forms of cerebral automatism. Two chapters are devoted also to phantasms. The general impression made by a perusal of the book is that the author has devoted himself to presenting as strong as possible a plea for the more occult side of the phenomena with which he deals. He is evidently a believer in the existence of something not quite yet known to our philosophy, and not recognizable or measurable by the ordinary methods of scientific investigation. He believes with a very positive conviction that there is some force or faculty yet unknown at the back of all the curious phenomena which go by the names of telepathy, clairvoyance, etc. The collected evidence in favor of their being some kind of ultra-material energy is sifted and presented in a very interesting and scholarly manner. The author is evidently quite familiar with the objections to the standpoint which the spiritualist takes, and the book is especially valuable for presenting all the evidences for the immaterial side. Perhaps the most tangible subject, which the promoters of psychic research have investigated, and the one as regards which scientific skepticism has dealt most kindly, is that of thought-transference. Regarding this point Dr. Mason says that he does not claim that thought-transference is established beyond all possibility of doubt or cavil, but that the views of those who believe in it are based on solid facts, and that the interpretation made of them is reasonable. The data which are presented in connection with these statements are certainly very striking and interesting.

Dr. Mason's conclusions regarding hypnotism are conservative enough, for he only states that he believes in the reality of the hypnotic condition, in the increased suggestibility of a hypnotized subject, and in the usefulness of hypnotism in therapeutics.

As regards clairvoyance, the author avers that there is abundant evidence of visual perception by some specially constituted persons independent of the physical organs of sight. If the various anecdotes and variously quoted incidents bearing upon this point could all be considered quite true and not coincidences, we might be disposed to agree with the author. The unfortunate fact in this, as well as in nearly all the other questions which are dealt with by Dr. Mason, is that they cannot be submitted to actual test under given conditions like the physical and chemical phenomena in the laboratory. All accepted scientific facts, except, perhaps, the broader deductions of philosophy, are believed in by cautious men because they can be absolutely demonstrated at any time with the proper surroundings and instruments. Until those who are enthusiastic believers in the ultra-physiological manifestations of the body can bring their patients and demonstrate the facts, they can hardly find fault at the presence of a general skepticism. We confess to a feeling of some sympathy, however, with the workers in the line which Dr. Mason is following, and we believe that such work should be received with sympathy and attention, especially when it is put forth with so much sincerity and scholarship as is shown in Dr. Mason's work.

C. L. D.

THE
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*Twenty-third Annual Meeting, held at St. John's Parish Hall
Washington, D.C., May 4th, 5th, and 6th, 1897.*

The President, Dr. M. A. Starr, in the chair.

LITTLE'S DISEASE; SHALL WE RETAIN THE
NAME?

By B. SACHS, M.D.,

Professor of Mental and Nervous Diseases in the New York Polyclinic, etc.

The various types of cerebral spastic palsies in children have received so much attention during the past five or six years that it might seem quite superfluous to continue the discussion of them; but the subject is one that grows from year to year. Few diseases of the central nervous system have presented as many puzzling problems. Freud's masterly monograph,¹ exhaustive yet not prolix, is the best evidence of the importance to which these various types of disease have attained. On the majority of points under discussion a unanimity of opinion has been established, but of late German and French authors have created some confusion by insisting that Little's disease shall be recognized as a distinct clinical entity. At the sametime

¹Die Infantile Cerebrallähmung, von Dr. Sigm. Freud. Nothnagel's Specielle Pathologie, etc., Band IX. Vienna, 1897.

English and American writers are taunted with the remark that they appear to have remained in ignorance of Little's views, though they might have been expected to have known them far better than the Continental authors of the day. One of the latest German authors,² states that "English and American authors make no distinction between Little's disease and double cerebral palsies; little do they care whether these two diseases are to be differentiated from one another or are to be considered identical." This remark is evidently inspired by a hasty reading of Freud's monograph. The latter author finds it strange that in spite of Little's excellent descriptions "no distinction is drawn in Anglo-American literature between general rigidity and bilateral cerebral palsies." "German and French authors are guilty of another error," Freud continues, "in failing to recognize the gradual transition between rigidity and paralysis".... "Little's hemiplegic spasmo-paralysis is merely a spastic hemiplegia."³

The apparent ignorance of English and American authors is accounted for by the fact that they soon recognized that there was no radical distinction between the types described by Little and the cerebral diplegias which we have learned to know in every detail. Osler⁴ in his monograph, published in 1889, recognized Little's merits, for he writes that he (Little) has contributed more than any one to the subject, and to him we owe in great part the accurate knowledge of the relation of the cases (of bilateral spastic hemiplegia) to abnormal parturition. In France, Osler adds, the cases of spastic rigidity are sometimes called Little's disease (p. 57). In my first study⁵ of cerebral palsies with Peterson, and in later writings, I have recognized Little's etiological group; it cannot,

²Grosz: *Archiv f. Kinderheilkunde*, 1897.

³Loc. cit., p. 23. Foot note.

⁴The Cerebral Palsies of Children. Philadelphia, 1889.

⁵Sachs and Peterson. *Journal of Nervous and Mental Disease*, May, 1890.

therefore, be fairly said that any of us was ignorant of Little's contributions; but, knowing them, we did not feel bound to indorse the conclusions which others have drawn from his writings; nor did we think it proper to establish "Little's Disease" as a clinical entity. There is some satisfaction that, in spite of the various insinuations that have been made, Freud has adopted our own point of view. His words are (p. 227): "We are forced, therefore, to the conclusion that Little's disease (so-called) cannot be accepted as a clinical entity, and the forms described under this heading must be classed with the diplegic types of infantile cerebral palsy." In spite of this concession, this author thinks it necessary, however, to retain the term "Little's disease" for those cases in which Little's etiology is a prominent characteristic, and in which the clinical symptoms are in agreement with Little's description. Freud's position is not logical, and it will not, I fear, help to clear away the confusion that has arisen.

Among French authors there is also a tendency to depart from what I must consider to be the rational interpretation of these various spastic types of infantile palsies. Brissaud,⁶ for instance, defines Little's disease as "a congenital and spasmodic paraplegia of all four extremities, more marked in the lower, and affecting more particularly children born before term, characterized by convulsive phenomena or intellectual defects, and susceptible, if not of a complete cure, at least of a progressive improvement." Brissaud has a special series of cases in mind which are familiar enough to all of us as spastic diplegias with slight mental involvement. He considers them to be of cerebral origin, and accounts for the preponderance of the affection in the lower extremities by insisting that it is very likely that the region of the paracentral lobule, whence issue the pyramidal fibres destined for the lower extremities, has the poorest blood supply of all cortical areas. If the cor-

⁶Leçons sur les Maladies Nerveuses. Paris, 1895. P. 110.

tical circulation is slower in the paracentral lobule than in all the other areas of the Rolandic region, this will suffice to explain the marked arrest in development of the pyramidal fibres going to the lower extremities. In passing we may note that Raymond⁷ has in one of his latest publications placed himself in line with Freud and the American authors; he recognizes various types of spasmodic spinal paralyses, but thinks that the lines of demarcation are purely artificial. Van Gehuchten⁸ in a series of articles published in 1896, entitled "*Faisceau pyramidal et Maladie de Little*," has given a very ingenious explanation for certain cases of congenital spastic palsy, but has created some confusion by considering the spastic palsies occurring in children *born before term* as the sole representative of Little's disease, and by insisting that if there is any one thing that is characteristic of Little's disease, it is the complete absence of paralysis.

Availing himself of Flechsig's investigations, Van Gehuchten attempts to show that in the newborn child at full term the white fibres of the pyramidal tracts have not yet acquired their myelin sheaths, and, furthermore, that in children born at seven months, the pyramidal tracts do not yet contain axis cylinders, at least below the medulla oblongata; from which he draws the conclusion that the cases of "Little's disease," so-called, are due to defective development of the lower portion of the pyramidal tracts.

If we were to ask in consideration of the preceding summaries, What is Little's disease? the only just answer would be, whatever an author pleases to make it. The cases described by the various writers have not even a common etiology, for in the cases described by Van Gehuchten, for instance, the asphyxia neonatorum, to which Little attached the greatest importance, plays no rôle whatever.

⁷Maladies du Système Nerveux, etc. Paris, 1894. P. 413.

⁸Journal de Neurologie et d'Hypnologie, June 5. 1896.

Before we decide whether or not we shall reject the designation "Little's Disease," let me give you briefly a few extracts from the English author's writings, and I do this all the more willingly as it is evident that he has been quoted so often at second hand that it would be a mere matter of justice to revert to the original article. "I showed," says Little,⁹ "that premature birth, difficult labors, mechanical injuries during parturition to head and neck where life has been saved, convulsions following the act of birth, were apt to be succeeded by a determinate affection of the limbs of the child which I designated, spastic rigidity of the limbs of new-born children, spastic rigidity from asphyxia neonatorum, and assimilated it to the trismus nascentium and the universal spastic rigidity sometimes produced at later periods of existence." Again he says: "I am justified in regarding the dissections of Hecker and Weber as confirmatory of the opinion . . . that asphyxia neonatorum through resulting injury to nervous centres is the cause of the commonest contractions which originate at the moment of birth, namely more or less spastic rigidity and sometimes of paralytic contraction." Please note this reference to paralysis. The existence of such is also insisted upon again and again when he refers to the greater weakness of muscles of the dorsal aspect of the trunk, to the muscles of speech commonly involved. He states especially that mental changes occur from the "slightest impairment which the parent unwillingly acknowledges and fails to perceive up to entire imbecility." He also believes that the spastic rigidity which follows asphyxia at birth is due to lesion of the spinal cord and not to lesion of the brain or medulla oblongata; that from some cause this nervous centre suffers most often from the asphyxia, or least frequently recovers its integrity. In view of these conclusions of Little, it is well to note that he records but two autopsies in more than sixty cases,

⁹Transactions of the London Obstetrical Society, Vol. III., 1862.

and that in these two there were distinct cerebral lesions. In one of these cases it is stated that the cord was not examined. Little, evidently, disregarded the post-mortem results and built up his views of the spinal origin of these spastic troubles on the clinical resemblance between these cases and those of spastic paraplegia in the adult. A careful reading of Little's articles leads to the following conclusions:—

1. He was struck by the prominence of rigidity and spastic contractures, but he acknowledges the occurrence of paralysis. On one of the two plates accompanying his article he pictures a child which he describes as "more paralytic than spastic."

2. The lower extremities are generally more affected than the upper, but this list of cases includes children with hemiplegia, with complete and incomplete diplegia, and some with paraplegia.

3. He ascribes these spastic palsies to difficulties during parturition and, above all, to asphyxia neonatorum. Moreover, it is particularly to be noted that in children prematurely born he ascribed the trouble not to defective development of tracts (of which he could have known almost nothing), but to the asphyxia which often occurs at the moment of birth when the change is made from placental to pulmonary circulation.

If we compare Little's contributions to this subject with the German and French writings previously alluded to, it becomes evident that his views have been considerably distorted, or at least modified, and that a number of recent investigators have tried to make the conclusions of more recent studies harmonize with views expressed fully thirty years ago. I have as much veneration as any one for the good work done by our predecessors, but we must not allow such veneration to be the cause of incalculable confusion. The researches on infantile cerebral palsies, published both here and abroad during the last

decade, have shown that the distribution of the paralysis, whether hemiplegic, diplegic, or paraplegic, was not a sufficient basis for differentiation; that such differences as existed were due to the varying extent to which one or both motor tracts were involved, and that it was wiser to divide all cases of spastic palsies in children into three large groups. First, those due to prenatal causes, including defective development; second, those due to difficulties during parturition; and third, the acquired cerebral palsies coming on during the first few years of life. This classification upon which I have insisted in previous writings, has been indorsed by Freud in his latest monograph. With the acute cases we are not concerned at the present time, but the groups one and two include cases like those described by Little, and the question therefore is, whether we shall surrender the advance made by establishing these separate groups, or whether we shall cling to this division and surrender the term "Little's disease."

To corroborate the impressions formed in former years, I have asked Dr. Onuf to examine the records of my clinic during the past few years, and to tabulate once more the "congenital," or more properly speaking, the "prenatal" and "birth" cases, with a view to indorsing or refuting the various doctrines which have been more recently promulgated. Fifty records were examined; of these eight were rejected, because some of the data had not been obtained with sufficient accuracy.

In these tables only such facts are given as bear upon the subject immediately under discussion.

From the subjoined table we may infer that premature birth, instrumental delivery, protracted labor, asphyxia, at the time of birth—any one or several of these conditions combined may be the cause of spastic hemiplegia, diplegia or paraplegia. Asphyxia neonatorum is an important etiological factor, but it does not overshadow all other causative agencies, as Little thought. Paraplegias cannot

SUMMARY OF 42 CASES OF PRENATAL AND BIRTH' PALSIES.

SEX & AGE.	TERM.	MANNER OF BIRTH.	FORM OF PALSY.	MENTAL CONDITION.	REMARKS.
1. F. 16 y.	Full.	Labor tedious, no instruments.	Spastic diplegia.	Excellent.	Mother kicked during 7th month of pregnancy; no convulsions in child.
2. F. 3 y.	Full.	Asphyxia.	Spastic diplegia, convulsive twitchings of both sides of face.	Fair, but no special development.	Mother had pneumonia at time of birth, dying five days later.
3. M. 3 y.	Full.	Difficult labor, asphyxiated (one hour).	Paraplegia.	Feeble-minded.	Marked hydrocephalus.
4. M. 3 y.	Full.	Slow labor.	Paraplegia, right hand defective.	Fair, has begun to speak.	Four convulsions at age of 2 years, beginning in right hand.
5. M. 4 y.	7 ms.	Labor lasted two days, not difficult.	Paraplegia, cross-legged position.	Imbecile.	Child weighed 3 pounds at birth, left arm weak, converging strabismus.
6. F. 3 y.	Full.	Instrumental delivery.	Hemiplegia.	Feeble-minded.	Microcephalus, slight prognathism, gothic palate.
7. M. 6½ m.	Full.	Protracted labor, instrumental delivery.	Spastic paraplegia.	Apparently good.	Frequent general convulsions.
8. M. 12 y.	Full.	Instrumental delivery, tedious labor.	Cross-legged progression, paraplegia.	Feeble-minded.	Cranial deformities, poor teeth, occasional convulsions.
9. M. 4½ y.	Full.	Protracted labor, instrumental delivery.	R. hemiplegia.	Fair.	Associated movements.
10. M. 2½ y.	Full.	Forceps, asphyxiated.	Spastic paraplegia.	Idiocy.	Convulsions when 8 days old, none since.
11. M. 16 m.	Full.	Short labor, asphyxiated.	Spastic diplegia, cross-legged position.	Backward (?).	Did not cry until 12 days old.
12. M. 3 y.	Full.	Normal labor.	Spastic paraplegia.	Good.	Six weeks after birth convulsions, no epilepsy, hands weak.
13. F. 9 y.	Full.	Normal, but no movements felt some weeks before birth.	Hemiplegia.	Imbecile.	Epilepsy since age of two years.
14. F. 3 y.	Full.	Difficult labor, shoulder presentation, forceps.	Spastic diplegia.	Deficient.	Convulsions twice in two years.
15. F. 6 y.	Full.	Sudden, after severe fright.	Spastic hemiplegia.	Fair.	Frequent convulsions of right arm.
16. M. 5 y.	Full.	Normal.	Spastic paraplegia.	Good, in spite of distinct microcephalus.	Two convulsions at age of 3 years, congenital strabismus.
17. M. 3 y.	Full.	Protracted labor.	L. hemiplegia.	?	Convulsions during 48 hours after birth.
18. F. 5 y.	Full.	Instrumental delivery.	Spastic diplegia.	Fair.	No convulsions.

SEX & AGE.	TERM.	MANNER OF BIRTH.	FORM OF PALSY.	MENTAL CONDITION.	REMARKS.
19. M. 2 y.	7 ms.	Fright hastened birth.	Spastic paraplegia.	Fair. began to talk at 15 mos.	
20. F. 3 y.	Full.	Asphyxia, first child.	Spastic paraplegia.	Very defective, microcephalus.	Spasm for ten days after birth.
21. F. 3½ y.	Full.	Normal, first child.	Spastic hemiplegia.	Defective.	No speech development.
22. M. 4 y.	Full.	Midwife brought on labor.	Spastic hemiplegia.	Good.	No convulsions.
23. M. 3 y.	7 ms.	Easy.	Spastic paraplegia.	Defective particularly in speech.	No convulsions.
24. M. 5 y.	Full.	Normal.	Paraplegia.	Fair, microcephalus, convergent strabismus.	Convulsions at age of three years.
25. F. ?	Full.	Instrumental delivery.	Spastic paraplegia.	Fair.	Cross-legged progression, no convulsions.
26. M. 7 y.	Full.	Difficult.	Spastic diplegia.	Deficient, no speech development.	Cross-legged progression, associated and choreic movements.
27. F. 3 y.	Full.	Asphyxia, first born.	Spastic paraplegia.	Deficient, no speech development.	Microcephalus, convergent strabismus, convulsions ten days after birth.
28. F. 3 y.	Full.	Normal.	Spastic hemiplegia.	Deficient.	No convulsions.
29. M. 3 y.	Full.	First born, asphyxia, instrumental delivery.	Spastic diplegia.	Defective.	Convulsions almost daily.
30. F. 2½ y.	Full.	Difficult labor, asphyxia, instruments.	Spastic diplegia.	Defective.	Convulsions daily.
31. M. 6 y.	Full.	Instrumental delivery, tedious labor.	Spastic hemiplegia.	Feeble-minded, microcephalus.	Defective speech.
32. F. 6 y.	Full.	Tedious labor, asphyxia.	Spastic diplegia.	Fair.	Convulsions about 3 times a month.
33. M. 3½ y.	Full.	Normal.	Spastic paraplegia.	Good.	Complete hydrocephalus.
34. M. 3 y.	Full.	Asphyxia at birth.	Paraplegia.	Good.	
35. F. 2 y.	Full.	Tedious labor, instrumental delivery.	Spastic paraplegia.	Bright.	
36. M. 19 m.	Full.	Slow labor, asphyxia.	Spastic diplegia.	Defective.	No convulsions.
37. M. 2½ y.	Full.	Normal.	Monoplegia (right arm)	Good.	No convulsions. R. k. j. increased.
38. M. 17 m.	Full.	Normal.	Spastic paraplegia.	Fair.	Got worse after whooping cough at 6 months.
39. F. 10 y.	Full.	Asphyxia.	Spastic hemiplegia.	Good.	Convulsions at age of 2-3 years.
40. M. 2½ y.	Full.	Asphyxia, one of twins.	Paraplegia with great rigidity.	Good.	No convulsions.
41. F. 16 m.	Full.	Tedious labor.	Spastic paraplegia.	Fair.	Convergent strabismus, microcephalus.
42. F. 3½ y.	Full.	Normal.	Spastic paraplegia without rigidity.	Defective.	No speech; no convulsions.

be attributed solely or even chiefly to an asphyxiated condition at birth, and it is entirely unwarranted to suppose that congenital spastic paraplegias are always, or chiefly, due to defective development of the pyramidal tracts in children born before full term, as Brissaud and Van Gehuchten would have us believe; for of the 42 cases of congenital cerebral palsies here recorded only three occurred in children born before full term.

If we are to give the name of Little's disease to conditions of spastic paraplegia in children prematurely born, we should be leaving out of account the very cases which Little had in mind, namely, spastic diplegias and paraplegias due to asphyxia neonatorum; and if we were to include under this term only those cases in which there is some form of palsy due to abnormal delivery, we should exclude the important series of cases in which injury to the mother during pregnancy, some intercurrent disease of the mother, or a distinct hereditary factor, had exerted its influence over a child born at full term. In 23 of these 42 cases there was some abnormal condition of labor; and in seven others the child was born asphyxiated, although all other conditions were favorable. These facts seem to me fully to justify the conclusions drawn from previous studies.¹⁰

I am willing to concede that the rigidity is sometimes more striking than the paralysis, and can endorse the views of Dejerine that the difficulty in locomotion seems (at times) to be proportionate to the spasticity, but it is equally evident that the rigidity is almost invariably associated with some degree of paralysis. It is in entire keeping with our modern conceptions of the anatomy of the central nervous system that these conditions should now and then be due to defective development of the brain and of those tracts which descend from the brain into the spinal cord, and such defect of development may occur

¹⁰Sachs. Volkmann's Vorträge, 46, 47. 1892. P. 449, *et seq.*

in children born at full term. This defect may be noticeable at birth, but if the arrest of development is not so marked, the clinical symptoms may not become apparent until some months or even years after birth. I cannot accept Van Gehuchten's explanation for the entire series of cases that are due to defective development, nor do I wish to separate the spinal portion of the pyramidal tract from its cerebral connections; and I believe that we must endeavor to enlarge our horizon by insisting that all cases with distinct defect of the cerebro-spinal motor system shall be brought under one heading, and that it matters little for purposes of classification whether the clinical symptoms in a given case be due to a porencephaly involving cortical motor areas, or to a defect in the development of the spinal portion of the pyramidal tract.

Van Gehuchten's explanation is not entirely satisfactory for several other reasons. First, it could not possibly explain the large number of congenital spastic cases that are associated with marked intellectual defect, and you may possibly remember that such defects have been shown by Peterson and me¹¹ to occur in 71 per cent. of the diplegic, and about 82 per cent. of all paraplegic cases. Secondly, if this theory is to be accepted for the purely spastic cases without mental involvement, it would be difficult to explain why all seven months' children are not affected with spastic paralysis. In some children we must suppose that, although, they come into this world before time, the pyramidal tracts undergo normal evolution in the earlier period of extra-uterine life. In spastic children the normal evolution does not take place. Why this should be so, neither Van Gehuchten nor any one else is as yet able to explain. But I would not detract from the significance of the Belgian author's views, for I believe that he has given us the key to the relation between some of these congenital spastic palsies, and the hereditary spastic palsies

¹¹ Journal of Nervous and Mental Disease, May, 1890.

epileptic symptoms, might well be explained on the theory that there was a meningeal hemorrhage at birth into the spinal canal. These cases, however, seldom come to autopsy. In fact, I do not know of an autopsy on such a case in literature. But the name Little's disease has still its application to this restricted class of cases.

Dr. F. W. Putnam.—It seems to me that these developmental symptoms—or the lack of development—from deficiency of certain portions of the nervous system, will vary always in their symptomatology quite as a matter of accident. For instance, take the symptomatology of the disease called Friedreich's ataxia. At present I have three cases under consideration; the eldest patient is six years old, and has paralysis of both extremities, absence of knee jerk, and lack of mental development usually belonging to his age. His sister also has the absence of knee jerk, and also headache sometimes, but has not the same amount of paralysis; she is able to walk with a waddling gait, but falls down very easily. That child is four years old, and is able to walk and stand, and her mental development is better than that of her brother, who is six years old. She is able to talk better than he can, and carries out an order or command better than he does. The child next youngest is about two and a half years old. It also has no knee jerks; has slight nystagmus; has not yet learned to creep; and does not show the usual mental development of a child of its age. Curiously, it appears as if there were a family influence there; an etiologic factor. The mother states that her pregnancies were all marked by very long and prolonged nausea; that for the first seven months she nearly starved to death, as she expressed it. Whether or not that had any influence upon the nutrition of these children, or the development of their nervous system, is of course theoretical. It seems to me that we can see the phenomena of Friedreich's ataxia combined with symptoms indicating lack of cerebral development. There is a great deal of room for the classification of these defects manifested through the disturbance of the various sensory phenomena.

Dr. Sachs.—I do not think Dr. Peterson can disagree with me at this late date, as I do not disagree with him. I was a little reserved in my statements in regard to the pathology of these conditions, and inasmuch as Dr. Peterson has suggested the possibility of a difference between us, I will simply say that I am inclined to think that some of these spastic palsies which we thought were cerebral may possibly be due to a spinal defect. But surely a large number of these cases are associated with defective mental conditions, and I must conclude that in them there is either an association of a cerebral with a spinal condition, or that the paraplegia was after all due to a cerebral lesion. The absence of the mental defect in some pa-

tients can be explained by the fact that in those cases there is a strictly localized cerebral lesion, which need not necessarily be associated with any mental defect or any mental symptoms whatever. The change that has come over the views of most of the authors that have written upon these subjects of late is in the direction of conceding that there may be some cases that have a spinal origin; but I cannot yet believe that a majority of these cases are due to a spinal defect.

(To be continued)

ENCEPHALITIS AND LATE EPILEPSY. Jas. G. Kiernan, M.D. (Alienist and Neurologist, April, '97.)

Epilepsy occurring after 25, and that due to encephalitis, have points of special interest in common. Epilepsy following on the various forms of infantile encephalitis is more apt to be accompanied by trophic phenomena. It reacts badly to the bromides. Mental symptoms replace under the bromides the convulsions. The tendency to impairment of the circulatory innervation of the extremities is increased. The bromic dermatoses appear with great frequency. A nocturnal mental type, resembling somnambulism, takes the place often of the convulsion. Encephalitic epilepsy, while in many cases possibly Jacksonian at the outset, but too often becomes an epileptic constitution with all the phenomena of idiopathic epilepsy. After the age of 25, and most frequently between 35 and 40, in persons with no decided neurotic heredity, and in most of whom lues can be excluded, occurs an epilepsy which resembles that from encephalitis. As a rule, in these cases there has been a precedent period of nervous exhaustion attended by vertiginous states as its later development. These states are often preceded or followed by anomalous sensory disturbances compared by the pt. to "waves." There is a loss of, or dazed, consciousness, with or without motor explosions. Some of these states, even when with consciousness, are attended by localized jerkings of groups of muscles. All of these phenomena are clearly due to toxins resultant on nerve exhaustion. Normally the toxins produced in the body are eliminated by various channels. When any of these emunctories are interfered with, the phenomena of auto-intoxication appear. The alterations are peculiarly suited for treatment of states due to toxin. By destruction of the toxin through stimulus of hepatic action and elimination, they prevent its accumulation, and the resultant phenomena. The alkaline bromides do not seem to exert this influence. Of late, metallic bromides have been (it is claimed) united in two compounds, the liquor arsen. auri, and liquor arsen. auri et hydrarg. These alternated weekly give, in the types mentioned, undeniably excellent results.

FREEMAN.

Original Articles.

AN ANALYSIS OF THREE THOUSAND CASES OF MELANCHOLIA.

By S. WEIR MITCHELL, M.D.,
of Philadelphia.

While studying melancholia I observed that certain cases seemed to be apt to relapse in the spring or summer, and I desired to learn if this tendency applied to the onsets of such cases as come but once in a life, or to those which repeat themselves at irregular intervals far apart in time.

With these points in view I searched the literature widely, but could obtain no satisfaction beyond vague, general statements. I asked Dr. John B. Chapin, of the Pennsylvania Hospital for the Insane, to let me have a list of dates of admission for melancholias, and I also requested Dr. Robert Chase, of Frankfort, and Dr. Henry Hurd, of Johns Hopkins, to give me their views as to the matter. While the results thus obtained are interesting, they show the need for further study.

Dr. Chase writes:—"In your reply to your letter of the 24th, instant, I hasten to say that in 2,220 admissions at Norristown, covering a period of five years (of whom about 30 per cent. were subjects of melancholia), a larger number were received in the months of April, May and June, and again in August, than during any other months of the year. The smallest number were admitted in October, November and February. This, I think, conforms quite closely to general statistics on the subject.

"The result of my observation in seasonal recurring melancholia is that relapses are more prevalent in the spring, reaching the maximum in June. This also is in

confirmation of statistics respecting suicides, that the wave of accession reaches floodtide in June."

Dr. Hurd writes:—"My impression is that at the North we are more apt to get melancholias in the winter, especially when the vitality of feeble patients has been depressed by unexpected or long-continued cold weather. I remember in a general way when I was in Michigan we always expected a large number of cases of melancholia in the months of January and March.

"As to recurrence of melancholia, I am not able to speak with so much certainty. Recurrent cases do not seem to follow any special law, but develop irregularly."

I give next Dr. Chapin's statement of 680 dates of admission of melancholias to the Pennsylvania Hospital for the Insane:—

TABLE I.—Admissions covering a period of thirteen years.

	Men.	Women	Total
January	24	37	61
February	21	39	60
March	28	25	53
April	24	27	51
May	18	41	59
June	25	44	69
July	23	35	58
August	20	36	56
September	22	37	59
October	20	32	52
November	19	32	52
December	16	34	50
Total.....	260	420	680

Here the numbers teach us a little, as a glance at the figures will show. June seems to offer the largest number. So many conditions in the various social classes determine the dates of admission that I am puzzled how usefully to employ these statistics.

Seeing that this table gave no distinct results as to the influence of seasons, I asked permission of Dr. Chapin to have studied the dates assigned for the time of origin, feeling that hospital notes might afford a reasonable approach to accuracy in fixing the time of beginning of the neurosis.

They are, of course, liable to many innocent misstatements, and must, I fear, be always open to criticism; but less and less so, as increasing care is given to the matter by the asylum physicians.

Dr. Chapin kindly permitted my assistant, Dr. Pearce, and my stenographer to examine and collect from his books 3,037 cases diagnosed as melancholias; 1,780 were women, 1,257 were men. The time covered lies between 1841 and 1896, as shown in Table II.

TABLE II.—Months of origin of melancholias.

	1257 Cases Male.	1780 Cases Female.	Combined Cases.
January	79	127	206
February	116	153	269
March	120	151	271
April	118	168	286
May	104	156	260
June	100	166	266
July	106	145	251
August	86	162	248
September	109	115	224
October	93	137	230
November	104	145	249
December	122	155	277
Total.....	1257	1780	3037

In this list the largest number of male cases is for December, but the numbers vary little except for August, which falls to 86. The largest number of female cases is in April, the smallest in January.

In Table III. the percentages are calculated, and in this shape the comparison is more easily seen.

TABLE III.—Percentage of occurrence of melancholias by months.

	Male Cases, 1257.	Female Cases, 1780.	Combined, 3037.
January	6.285	7.135	6.783
February	9.229	8.595	8.857
March	9.547	8.483	8.923
April	9.338	9.438	9.417
May	8.228	8.704	8.567
June.....	7.956	9.326	8.758
July	8.448	8.146	8.251
August	6.845	9.101	8.166
September	8.625	6.461	7.385
October	7.399	7.697	7.573
November	8.288	8.146	8.199
December	9.702	8.708	9.121

April gives, of the total men and women, 9.417 per cent., and December comes next—9.121 per cent. Too much may be made of such a table. It serves to make clear, however, how very small is the difference of time of origin. The late winter and early spring months seem to offer the most cases. I confess myself surprised at the want of more differing results for the several seasons.

While thus employed my assistants took note of some other matters which proved to be quite worth this trouble. One was a table, which I do not print, of the causes assigned as parents of melancholia. This table satisfies me that some very carefully planned measure is needed to bring this matter into useful form.

I am told that the Psychological Society of North America has a committee thus engaged, and, with this knowledge, to criticise would be unnecessary and without useful end.

An effort was made to state the duration of melancholias in 422 cases where the time was noted, and Table IV. shows the continuance of the disorder previous to admission, as stated by the friends. This statement, as it stands, is a strange record, and appears to show how many cases remained at large after the onset of the disease before they were placed in the institution. Possibly a large percentage had been in other asylums, and the table has no serious value.

TABLE IV.—Duration of attack at time of admission.

	MALE.	FEMALE.
One month or over.....	1	0
Six months.....	2	2
One year.....	88	83
Thirteen months.....	0	1
Fifteen months.....	0	1
Eighteen months.....	6	8
Two years.....	44	51
Two and one-half years.....	8	1
Three years.....	26	19
Three and one-half years.....	2	1
Four years.....	6	16
Five years.....	9	8

Six years.....	6	8
Seven years.....	0	1
Eight years.....	3	1
Nine years.....	1	0
Ten years.....	3	5
Thirteen years.....	0	1
Fifteen years.....	2	0
Sixteen years.....	1	0
Twenty years.....	2	0
Twenty-two years.....	0	1
Twenty-four years.....	0	1
Twenty-six years.....	0	1
Thirty years.....	1	0
Thirty-one years (case of trauma).....	1	0

TABLE V.—Comparative percentage of cases by decades.

	MALE.	FEMALE.
Under twenty years.....	3.7	6.2
Between twenty and thirty years.....	26.8	26.4
Between thirty and forty years.....	27.6	27
Between forty and fifty years.....	20.2	21.4
Between fifty and sixty years.....	15	14.2
Between sixty and seventy years.....	5.4	4.2
Over seventy years.....	1.3	.6

This comparison of ages is most striking, and the correspondence in age in the two sexes is well worth attention. The cases in both sexes might have been better grouped, so as to bring out the influence of the menopause; nevertheless, even as at present arranged it is valuable, and confirms statements from other asylums; also it leads to the observation that in men the percentage between forty and fifty years is 20.2, and in women but 21.4. Or, if we take the years from fifty to sixty, the percentage of male cases would be 15, and of female cases 14.2. These two sets of figures seem to dispose of the idea that women are more liable to melancholia at or about this critical period. But in order to get at this even more precisely, I had the percentage calculated for the years from forty-five to fifty-five, with a result which shows practically no difference from the statement for the decade from forty to fifty.

TABLE VI.—Percentage of ages between 45 and 55 years.

Number of male cases.....	289
Average age, in years.....	47.5
Percentage	20.25

Number of female cases.....	354
Average age, in years.....	49
Percentage.....	19.8

Again, if we consider the whole table, it would appear that the time of greatest liability is for both sexes between twenty and sixty years, but under twenty it is relatively nearly double for women.

In further confirmation of the fallacy of attributing melancholia to the menopause, I append some statistics furnished by Dr. Ely Josselyn, of the Pennsylvania Hospital for the Insane.

NEW YORK STATE LUNACY REPORT, TABLE III., P. 526.

"From 1888 to 1894 there were 16,208 admissions, of which 7475 were women. Of this number, 282 cases were attributed to menopause—less than 4 per cent.

"In five asylums in Massachusetts: Worcester, Taunton, Northampton, Danvers, Westborough, we note the following:

"In 1895-'96.

"Worcester 292 admissions, of which twelve were attributed to the menopause—4 per cent.

"Taunton 209 admissions; three attributed to the menopause—less than 2 per cent.

"Northampton 94 admissions; two attributed to menopause—less than 3 per cent.

"Danvers 218 admissions; seven attributed to the menopause—less than 4 per cent.

"Westborough 276 admissions; four attributed to the menopause—less than 2 per cent.

"At McLean Hospital of 63 admissions two were attributed to the menopause—less than 3 per cent.

"In the Connecticut Hospital for the Insane, since its opening, in 1868, of 3607 admissions, 86 were attributed to the menopause—less than 3 per cent."

In the reference above it is clearly shown that of all insanities recorded in these institutions only about two per cent. were attributed to the menopause. If this obtains in general insanities, the question arises how large a factor the climacteric is in melancholias alone

Through the kindness of Dr. Moulton, of the Pennsylvania Hospital for the Insane, I append the following extract from the United States Tenth Census Report:

"Of the 27,105 cases of insanity tabulated by the Tenth Census, there was one male lunatic to 574 persons, and one female lunatic to 518 persons.

"While this would seem to contradict the assertion that more men are insane than women, it must be remembered that more men die of paresis than women. More women are found in hospitals than men, because not many female cases are fatal.

"In seven hospitals in Massachusetts (a State where there are more women than men in the general population) out of 63,525 people the commitment of men exceeded that of women by 605."

TABLE VII.—Number of cases having had prior attacks—672.

	MALE.	FEMALE.
One prior attack.....	168	277
Two prior attacks.....	38	99
Three prior attacks.....	18	28
Four prior attacks.....	11	15
Five prior attacks.....	3	4
Six prior attacks.....	2	0
Seven prior attacks.....	2	0
Eight prior attacks.....	2	0
Nine prior attacks.....	2	1
Ten prior attacks.....	0	0
Eleven prior attacks.....	0	0
Twelve prior attacks.....	0	0
Thirteen prior attacks.....	0	1
Total.....	246	426

I felt too doubtful of the value of these statements to think it worth while to work out the percentages. Females seem more liable to recurrence of attacks than men.

Of the number of cases under consideration (3,037) the age was given in 2,547 instances, and the averages are given in the following table:—

TABLE VIII.—Age of occurrence of melancholias.

Average age of males.....	37.2
Average age of females.....	36.25
Oldest male case.....	76
Oldest female case.....	78
Youngest male case.....	10
Youngest female case.....	12

Considering the very different condition under which the lives of the two sexes are passed, this close relation as to time of liability to attacks is interesting.

I thought some of the points made plain by these tables valuable enough to make it worth while to print them; but I have been cautious about inferences, and should like to see what I have attempted worked out on a larger scale by the publication of the records of a number of hospitals, and for all the distinct types of insanity.

I have the privilege of using my friend Dr. John B. Chapin's valuable comments on these tables. These relieve me from the need to say anything further; they are of remarkable interest. Dr. Chapin says: "I hardly like to venture any criticism, or even to make suggestions, about a work on which you have expended so much time.

"As for the table showing the seasons at which melancholias make their appearance, I think it is, as you say, negative of all results, except in showing that there is no 'seasonal melancholia.'

"As to the date of attack, the hospital and the physician preparing the certificates of admission depend mainly for the history of the case upon the friends of the patient, only some of whom are intelligent, and even these are often not correct observers. Therefore, the date of the insanity is usually fixed at the time some open manifestation takes place, and no account is taken of the incipency which may have been in operation for months. As your tables are the first contribution in the line of this inquiry, they must be accepted as of authority, although it should be remembered that they are based largely upon the admissions to a hospital receiving patients from a city population in the latitude of Philadelphia. The dates which make up your table are all that are accessible for this locality.

"I entirely agree with you about the small value the table of assigned causes has in this relation. I have never attached any importance to any table of this character

that I have seen published. It is usual to enter in the case-book the 'assigned cause' of insanity furnished by the friends as a part of the history of the case. It is not always the cause which the physician would assign. To estimate the cause of any insanity is such a complex operation that sufficient time cannot be given to the analysis of every case to make the results of value. For my own experience I have sometimes made a classification depending upon whether the disease was supposed to be recoverable or irrecoverable; whether acquired or due to organic degeneration.

"Table IV. is valuable as showing approximately how long such patients are at large before admission to an institution. It is useful, too, in giving some idea of the duration of the disease, and it is again the only information obtainable in this direction.

"Table V. I consider the most valuable contribution to knowledge which your paper furnishes. In the first place, you have established by Table II. that more men are sent to hospitals with melancholia than women, and it is true that more men become insane than women.

"The estimation of the occurrence of melancholia in the years between forty-five and fifty-five, Table VI., shows that the climacteric period, which all women dread who have a predisposition to insanity or to any neurosis, exerts but little influence upon this condition; an equal number of men and women become insane at this period. In this connection I may remark that the expectation that insane women are to recover at the climacteric period is equally fallacious. This fact, which I am not able to verify by statistics, is difficult to prove, but I am quite assured of its truth from observation, and you may as well explode the tradition as anyone else. The decades following fifty show an increased percentage of men, and this might be expected, as the known fact is that more men become insane than women. The facts shown by this table are worth all of the labor you have bestowed upon your paper."

CONSIDERATIONS ON FLECHSIG'S "GEHIRN UND SEELE."¹

By MARY PUTNAM JACOBI, M.D.,

I do not this evening propose to offer either a translation or a detailed exposition of Flechsig's essay, which has already become so celebrated. For not only do I take for granted that it is, in the original German, sufficiently familiar to the members of the Neurological Society, but Dr. Barker, for the benefit of the Philadelphia Neurological Society, has already made an English translation, which has appeared in the June issue of the *Journal for Nervous Disease*. Dr. Barker's translation, however, is completely uncritical, and it seems to me that room remains for some modest criticism of Flechsig's large and confident conclusions.

The essay in question contains: 1st, an interesting anatomical discovery; 2d, a physiological interpretation of this, and 3d, a philosophical inference deduced from this alleged interpretation. In this inference lies the gist of the title of the essay, and the conclusions which the author himself seems to find, at this time, the most interesting. In his earliest treatise in 1876, Flechsig announced the following laws as the main results established by his discoveries upon the unequal medullization of nerve tracts:

1st. The law of organization of central nerve fibres into systems.

2d. The principle of the systematic assumption of myeline sheaths by all the nerve fibres belonging to the same system.

3d. The principle of the systematic division of the

¹ Read before the New York Neurological Society, October 5, 1897.

central medulla based upon the separate medullization of different nerve tracts.

4th. The correlation of the epoch of its medullization with that of the first differentiation, the first distinct appearance, of any system of central nerve fibres. These laws are sufficiently important and interesting and, I think, have not been controverted. But now Flechsig brings forward another law entirely new, and which is no longer, like the others, a generalized summary of the facts. He now tells us that "tracts of different functional significance ripen at different times," and that this simple fact is the keynote not only to the whole philosophy of the nervous system, but to the entire problem of the relations of brain and mind.

These are the successive steps of the demonstration. The earliest tracts to be medullated are the earliest tracts to be formed. At the time at which the mass of the cerebral hemispheres is still gray, tracts in them which have become medullated must be the oldest, and on that account presumably the most capable of function. This was said in 1876. But twenty years later Flechsig seems to infer that nerve fibres are incapable of even an embryo function until they have assumed their myeline sheaths; and, conversely, that successive awakening to function of tracts of different functional significance can be traced by the chronology of their successive medullizations.

In the cerebral hemispheres the earliest fibres to become medullated are contained in certain sensory tracts—tracts which mediate tactile sensation.

Hence, tactile sensation is the first thing produced in the brain. But the brain exactly mirrors the mind. We must, therefore, infer that mind begins in a sensation, and that other things are added as other nerve tracts ripen. "More than ever have I the conviction," exclaims Flechsig in the preface to his published address, "that the brain as organ completely and entirely coincides with the manifestations of the soul, and that we are in a position to develop the

conditions of the same as of all other natural phenomena accessible to our knowledge." And again: "We define the soul as a function of the body, mental phenomena as phenomena of life; distinguished from others by the fact that they are accompanied by consciousness. Consciousness is an accompanying phenomenon of this special process; by no means a resultant of the same in a mechanical sense."

Having said this, Flechsig then proceeds to deduce mental phenomena from anatomical facts, without the slightest further reference to consciousness. His demonstration is suggested, however, not by the anatomy which he professes, but by the metaphysics which he professes to despise. It is plainly inspired by the famous "statue" hypothesis of Condillac. To analyse the faculties of man, this philosopher imagined a statue deprived of all faculties, and then added to it (not faculties, but) sensations, one by one. He first calculated the effect of a single sensation which should exist all alone and be excited by one object only, as a sensation of smell excited by a rose in a being who had absolutely no other capacities than this sense of smell. He then added a second sensation of the same kind, as the smell of a jonquil, and demonstrated to his own satisfaction that a being who could only smell, but who had the opportunities to smell a rose and a jonquil, could develop from these two sense experiences, comparisons, judgment, passions, abstract ideas. "All the faculties of the mind," declares Condillac, "are only transformed sensations." "The mind is quite passive when it experiences a sensation." To this doctrine, enunciated in 1746, by the French Abbé, the German anatomist tries to bring the support of the anatomical science of the nineteenth century.

But this attempt is open to many objections.

1st. Except on the teleological basis which Flechsig would probably be the first to reject, the function to be performed by a completed organ cannot be counted among

the efficient causes of its mode of development. Yet there is an illegitimate emphasis in Flechsig's declaration, "Tracts of different functional significance ripen at different times." Although he does not say it in so many words, he manages to convey the impression that the tracts ripen at given epochs, because of their functional significance; or that this guided the order of medullization. Mind must be generated by sensation, therefore sensory tracts must be capable of function before any other part of the nerve centres, or, at all events, the relative precocity of their development affords a striking confirmation of the philosophical theories to which Flechsig evidently had already adhered.

But when we examine what is the efficient cause of the development of nerve tracts we find it in conditions with which functional significance has nothing to do. In 1876 Flechsig made the remark, which he now seems to have forgotten, namely, "that the systematic development of nerve fibres must depend on the fact that the *cells* which produce the fibres ripen at different times."² The contrast between the epochs of medullization of afferent and efferent tracts, upon which Flechsig so greatly insists, and which, indeed, he greatly exaggerates, depends entirely on the fact that these tracts have different trophic centres.

At eight months of foetal life, the mass of the core of the cerebral hemispheres is still gray. At this epoch white streaks of medullization appear in the following localities.³ The posterior central convolution and the upper half of the anterior.

The upper levels of the external nucleus of the thalamus, where fibres are received through the superior peduncles of the cerebellum from the red nucleus of the tegmentum.

The posterior three-quarter of the globus pallidus.

² Leitungsbahn, page 209.

³ Gehirn und Seele, p. 62.

Edinger⁴ states the matter thus: "The tegmentum radiation (Haubenstrahlung) arises in the cortex of the superior parietal lobe and posterior central convolution, possibly in other cortical regions, reaches the internal capsule and partly passes under the thalamus to the cord, partly plunges into the inner segments of the lenticular body. Here are the fibres of the cerebral hemispheres which first become medullated. In a human foetus of from eight to nine months, these fibres appear as thin white streaks in the internal capsule, the rest of which is gray." "They occupy," says Flechsig in his essay on Localization, "the area in the upper half of the internal capsule, immediately behind the pyramidal tracts."

Flechsig rests the proof of the sensory character of the fibres in question upon two facts. First, that in the region where they enter the internal capsule, namely the posterior part of its knee, experimental section is followed by crossed hemianesthesia. This was, indeed, shown many years ago by Veyssi re and other pupils of the Charcot school. Second, that these same fibres may be traced through the lemniscus to the medulla and into direct connection with nuclei which are the final termini of spinal sensory nerves.⁵ This tract, however, is not the only sensory tract which extends between the cortex and the base of the brain. Edinger describes,⁶ under the name of the "cortical lemniscus," another bundle which passes from the cortex caudad to the anterior central convolution, and which ends in the ventral nucleus of the thalamus. The same nucleus receives lemniscus fibres from below.

Now Flechsig tells us, or leaves us to infer that this upper cortical portion of the lemniscus, if it be so considered, is *not* medullated at eight months. Indeed, Flechsig divides the sensory system into three parts, of which the

⁴Vorlesungen  ber den Bau der nerv sen Centralorgane. 1896.
P. 234.

⁵Edinger, loc. cit., p. 281.

⁶Loc. cit., p. 234.

first, as above described, begins to be medullated at eight months; but the second not till nine months, and the third not till several months after birth (*Local.*, pp. 24 and 25). According to Flechsig's description this second division of the sensory system constitutes a special system in the internal capsule which, like the first, passes out by the lateral nucleus of the thalamus, but dorsal to the first system. It passes to the cerebral cortex, partly to the paracentral lobule, and also to the foot of the first frontal convolution; partly bends inwards at an acute angle, and terminates along the entire length of the gyrus fornicatus; partly enters the cingulum and runs towards the cornu ammonis. Towards the epoch of birth, the foregoing bundles are joined by another which runs from the lateral nucleus of the thalamus, enters the unciform convolution, and finally reaches the subiculum cornu ammonis. Another bundle passes from the median centre of the Luys nucleus to the foot of the first and second frontal convolutions. The third system becomes medullated from one to several months after birth. It passes in the middle part of the internal capsule from the anterior part of the lateral nucleus, and runs directly to the foot of the third frontal convolution (*Local.*, pp. 24, 25). It is in a foetus nine months old, at term, that Edinger describes as medullated, fibres in the tegmentum, the lemniscus, the brachia (*binde-arme*), the posterior longitudinal fasciculus, and many fibres of the substantia reticularis.

Now at this epoch, nine months, or the moment of birth, Edinger finds a small bundle of medullated fibres also in the pes pedunculi, the motor tract, and Flechsig assigns the medullization of the pyramidal tract to the self-same time.

From this description it follows: First, that all sensory tracts do not "ripen" at the same times, but that an interval of about four months separates the period of medullization of the first division from that of the third.

Second, that the medullization of motor tracts is

exactly contemporary with that of several sensory tracts even if not with all. Yet Flechsig declares, "The motor tracts of the cortical sensory spheres develop without exception *after* the completion of the sensory tracts; this same law holds for all centripetal and centrifugal tracts of the cortex."⁷ This is called the "fundamental law of cerebral development." The law holds whenever the trophic centre of a centripetal tract is more completely developed than the trophic centre of a motor tract, and this is frequently the case. It does not hold below the level of the hemispheres, that is exactly at the points where an external medium can alone excite afferent impressions, thus where, if alone, the process of sensation must begin.

The trophic centre of the median and lateral lemniscus fibres is in the medulla oblongata. The trophic centre of the afferent fibres passing upwards to the convolutions in the knee of the internal capsule, is presumably the lateral nucleus of the thalamus. The trophic centre of the pyramidal fibres is unquestionably the cortex of the anterior convolutions and the parietal lobule. It is inevitable that this cortex gray matter of the brain mantle, the latest in philogenetic development, should complete its structural elaboration later than the gray matter of the nuclei of the thalamus and of the medulla which are fully formed so much earlier in the animal scale. It is correlatively inevitable that the tracts which originate in this latest-formed mass of gray matter should become medullated later than those which spring from the ganglionic masses at the base of the brain. As Flechsig said twenty years ago, "The different tracts develop probably in the same order in which the centres to which they belong develop."⁸ The indifference of function in the question of medullization is shown by Flechsig himself in his remarks upon the medulla oblongata. "Here," he says,

⁷Localization, p. 45.

⁸Leitungsbahn, p. 226.

"very early, groups of great cells of the formatio reticularis differentiate themselves, whose axis cylinder processes pass over in the fibres of the spinal anterolateral tracts; and these clearly centrifugal paths show well-developed medullary sheaths at a time when the sensitive roots are still deprived of them. Those motor cells and fibres are thus complete and capable of function at a time when the posterior roots are still embryonal. Hence, it is probable that for this lower part of the brain, not reflex, but automatic action is the primary form of the central function." Edinger (page 5) stating the same facts, gives to them an entirely different interpretation. "To the vital importance of the medulla for the existence of the animal," he says, "corresponds the circumstance that this part of the brain is the first to complete its evolution. At a time, between six and seven months of foetal life, when there is not a single medullated fibre in the entire frontal region of the brain, the cranial nerves originating in the oblongata have already assumed their medullary sheaths; and soon afterwards their central tracts, the tractus tecto-nucleares, become medullated. At this time also is medullated the posterior longitudinal fasciculus passing from the thalamus to the anterior columns of the cord."

According to this way of looking at the matter, in the sixth month of foetal life the ganglionic centres of the medulla begin to develop rapidly, in anticipation of the important functions they are soon to assume, and outstrip the spinal ganglia, the trophic centres for the posterior roots.

The sensory and motor roots of the spinal cord become medullated at about the same time, because their respective trophic centres, the spinal ganglia, and the masses of ganglionic cells in the anterior horns, complete their structural elaboration contemporaneously.

It may, perhaps, be said that the reference of medullization to the development of trophic centres makes no difference in regard to the inference to be drawn from it.

Whatever the cause, we must believe that a medullated nerve tract is capable of function as a non-medullated tract is not, both because its fibres must be older, and because the myeline sheath is essential to the function of the fibre. If, therefore, sensory fibres are invariably medullated first, they must invariably begin to function first, must in some way bring it about that sensations arise in the brain, or rather are passively stamped upon the brain before this organ is capable of any reaction. But it has been shown by the observations of Edinger, and even by the data furnished by Flechsig himself, that if a few sensory fibres are the earliest in the hemispheres to become medullated, the medullization of many others is anticipated by that of many motor tracts.

Further, the development of the myeline sheath is not directly an expression of the perfected function of the nerve fibre, but of the *luxus nutrition* permitted by complete elaboration of its trophic centre. There is no proof, and no probability, that the conducting functions of the nerve fibres begin suddenly at a certain stage of development. Rather that the germ of function appears coincidentally with the germ of structural elaboration. Oscillations of blood corpuscles begin early in the vascular channels of the embryo, and constitute the first organic effort at the circulation, which is destined later to become so impetuous. Similarly feeble, it may be presumed, would be the first nerve impulses—molecular oscillations to and fro—until, as in the blood vessels, such continuity of channel had been established as would permit complete circulation.

Van Gehuchten denies Flechsig's implied assertion that the myeline sheath of central nerve fibres is essential to the conduction of nerve impulses, because, as he points out, there are many cases in which, in the absence of this sheath, function is unquestionably performed. Throughout life all the fibres of the sympathetic system, the fibres of the olfactory nerve, the central and peripheral fibres of

the cerebrospinal system at both their origin and their termination, and all the protoplasmic prolongations of all nerve cells except in cerebrospinal ganglia, remain destitute of myeline. According to Lenhossek, in mice, the entire spinal cord is still non-medullated at birth, the first medullated fibres appearing in the anterior commissure on the third day after birth. In these same animals the cerebrospinal fibres of peripheric nerves only become medullated at some little time after birth. Yet mice begin to run about at least as early as many other mammalian animals, born with medullated cords. And viable children, born at seven or eight months of foetal life, move their limbs in a manner quite indistinguishable, except perhaps through degree of force, from movements of children born at term. The only condition indispensable to the function of nerve fibre is adequate anatomical connection between neurons. The myeline sheath simply facilitates the process of conduction, by securing better isolation of the fibre. Hence its importance for cerebrospinal nerves during their prolonged course beyond the limits of the neural axis, or even beyond the central cortex or spinal central gray matter. While within those limits the myeline sheath is evidently superfluous, for it does not exist, and perhaps might be imagined to even impede the extreme rapidity of communication required between ganglionic centres. Lenhossek thinks that the myeline is gradually deposited from the surrounding plasma upon the axis cylinder, and as in the case of all organic tissue elements, the functional activity of the fibre stimulates the attraction of the plasma and the formation of the sheath, which constitutes a species of *luxus nutrition*. Again, so far as we know, elementary nervous processes are always carried on in circuits, formed by an afferent channel, a central arc, and an efferent channel. Even the specific afferent impulses of the visual sense are followed by incessant motor responses through the ocular muscles. There is no precedent for the conception that definite afferent sensory impulses are transmitted to a

nerve centre, there to accumulate until the centre and its centrifugal tracts had become sufficiently developed for response. The statue of Condillac was a pure fiction, and a rather clumsy one at that. Dare we say the same of the laughter hypothesis of Flechsig?

On the supposition that embryonal conducting function begins in embryonal nerve fibres; and that the appearance of the myeline sheaths is not essential to function, but an incident to the nutrition of the developing fibre, the dramatic contrast between sensory and motor tracts "of different functional significance" disappears. The brain is not found to be absolutely passive at one time of its existence any more than at another.

It is curious to note that in his earlier book Flechsig expressly observes that the medullization of a nerve tract cannot be accurately determined by its white color. Myeline sheaths may be fully formed, and yet, until they have reached a certain size, the tract will remain gray. This, he tells us, is the case with the columns of Goll. Yet no more exact test than that of color seems to have been applied in tracing the medullization of nerve tracts in the hemispheres. And Flechsig's own data leave room for the possibility that myeline sheaths are formed at various points of the hemispheres, where as yet white streaks have not appeared.

Thus, as it seems to me, even the histological details described by Flechsig lack sufficient support, and still more his physiological inferences. But the philosophical conclusion which we are asked to accept on this tottering basis is really amazing. An afferent neural impulse, said to be transmitted to a nerve centre so imperfectly developed that it has not yet secured the medullization of its efferent tracts, is treated as a sensation. It is said to precede the first appearance of consciousness, and yet to be the corner stone upon which all consciousness is built.

But a sensation includes much more than an afferent neural impulse. At the very least it includes the central

excitation of gray matter in the brain cortex. It does not precede consciousness, for it constitutes one of the phenomena of consciousness, often of peculiar vividness. There may be, and there are many nervous excitations of which the individual remains entirely unconscious, but an unconscious sensation is a contradiction in terms. Consciousness corresponds to the total activity of the brain cortex, and necessitates the completion of its circuits. It probably depends upon much else besides, but certainly upon this. According to Meynert's picturesquely described scheme, partial revivals of different memory residues occur, coinciding with local affluxes of blood to different cortical areas; and thus the general stream of consciousness is reinforced at different points somewhat as a continuous murmur in the carotid receives systolic reinforcements. But until or unless the general stream exist, there can be no partial reinforcement, no individual phenomenon of consciousness, no sensation.

An afferent neural impulse is not a sensation, but expresses one form of the general susceptibility to external excitation, which is characteristic of all organized beings. Because, even before a nervous system is developed, such excitation is always followed by an organic reaction; so, when the nervous system begins to be organized, and external impressions are made upon afferent nerve tracts, the reaction is effected through efferent nerve channels. There is always a circuit, and the nervous reflex is, admittedly, the general type of nervous action at all levels of the nervous system. Flechsig professes to find additional proof of the reflex action of the hemispheres in his new data of the precocious medullization of sensory tracts. But there can be no reflex without the coincident function of centrifugal tracts, and this latter, according to Flechsig, is for many weeks in complete abeyance. The more Flechsig's theory on this matter is examined, the less it is found in agreement with anything which is known or admitted

about the correlation of function with organic structure. Nowhere else has it been suggested to dissect the complete function of a developing organ and assume that one-half of this function could be completely performed while the other half could not be. As well assume that there could be a systole of the heart without a diastole, an inspiration without expiration, glandular elimination without excretion, as to assume that afferent impulses could be carried in a nervous system totally incapable of efferent or centrifugal neural action. Still less plausible, if possible, is the assumption of a brain receptive to all external impressions, yet devoid of the power to react to these according to its specific individuality. By this scheme Flechsig has not succeeded in superseding Meynert's analysis of the gradual development of conscious volition, through a series of acts at first unconscious, but successively recorded on the cortex of a correlatively developing brain.

To effect a record of acts performed or of impressions received, a record that subsequent acts are able to revive, so that many may be simultaneously present in consciousness, and that the continuity of the stream of consciousness may be preserved—this seems to be the one function of the non-sensory areas of the brain cortex. In this way this marvelous structure specializes and intensifies the general power of memory, which is the fundamental attribute of all organic tissue. Consistently with the conception of one simple and uniform function to all the non-sensory areas of the cortex, we find, it is said, in these a uniform and relatively simple structure. And this is the chief anatomical distinction between the areas of the brain which constitute central expansions of nerves, the sensory areas, and the remaining portions which are not sensory. The latter are the so-called psychic areas of Flechsig, identical with the regions which, several years ago, were called by Broadbent "the superadded convolutions." "There is not only one seat of the soul," exclaims Flechsig, "there are at least two—the great anterior, and great posterior asso-

ciation centres." "The attribute of a soul-making function to the centres in question rests primarily on an argument of exclusion. The mind," says Flechsig in substance, "is a product of the brain. Such and such parts of the brain do not produce mind, but only sensory impressions or motor impulses. But as mind is clearly produced, it *must* originate in the action of the parts of the cortex which remain after subtracting the sensory areas. Therefore, these must be psychic centres." They are called also associating centres, because the genesis of mind is explained precisely on Condillac's hypothesis, by the simple addition, association, or blending together of two sensations.

"Any one sensation is not mind," declares Flechsig, although he had previously interpreted all his observations on the medullization of nerve tracts, as proving that mind was constructed from a sensation. Two sensations are not mind; but if two different sensory impressions meet in the region of the brain which is intercalated between the two sensory centres where such impressions are generated, then something new is formed by their association, and this something is an idea, a thought, a mental tendency, there is a piece of mind. In each psychic centre we can deduce the mental faculties which are there developed, by observing what sensory centres border on each side of it. The insula lies between the auditory and the motor speech centre: it is therefore the psychic centre for the faculty of speech. The parieto-occipital lobe is intercalated between the visual centre and the touch-motor centre, the somæsthetic area of Barker, constituted by the Rolandic convolutions. Hence the posterior psychic centre is the seat of faculties which may be considered as composed of the sense of touch and the sense of sight—faculties which tend towards scientific observation or æsthetic talent, as the case may be. Following this line of thought, we should expect to find the frontal lobes, intercalated between the somæsthetic centre and the olfactory sphere, to be the seat of faculties which

could be traced to a combination of the sense of touch and the sense of smell. But here the synthesis breaks down, and Flechsig, who localizes in the frontal convolutions the sentiment of personality, the sense of the *ego*, is much put to it to explain what share the olfactory sphere should bear in the genesis of this part of the mind. Unable to do so, Flechsig at this point abandons the strict application of his own theory, and deduces the sentiment of the *ego*, not from a combination between two sensory centres, but exclusively from the adjacency of one—namely the somæsthetic sphere in the central convolutions. The sentiment of the *ego* and of personality is said to arise as a natural sequence from the impressions of touch, which put the individual into connection with the outside world; and the impulses to movement, which give him control over it.

The foregoing exposition sounds wonderfully clear, and convincingly ingenious, until we attempt to analyze exactly what it means, and then the apparent clearness becomes veiled by the densest obscurity. The theory involves the following postulates:

1st. That sensations are not psychic phenomena. Flechsig defines psychic phenomena as vital phenomena distinguished from all others because accompanied by consciousness. Now, a sensation, to be such, must be accompanied by consciousness, often of peculiar vividness. Consciousness is not essential to organic susceptibility or to afferent neural impulses, but an unconscious sensation is a contradiction in terms. The first postulate, therefore, is false.

2d. That a mental act proper always implies generalization from two or more sensations, or from the memory residues of sensations. This statement raises the cardinal problems in philosophy, which are far beyond the province of this paper to discuss. It may be, however, admitted that for a certain class of mental acts the proposition is approximately correct.

3d. It is quite otherwise with the third postulate of

Flechsig's theory. According to this the generalization in question is affected by the propagation to an associative centre along associative fibres, of excitations originating in the sensory centres which lie on either side of it. Two specific excitations are said to combine with each other—thus a visual impression from the cuneus, with a tactile impression from the posterior central convolution—the combination taking place in the posterior psychic area of the parieto-occipital lobe.

But what possible meaning can there be attached to such a statement as this? What proof or precedent exists for the conception of a material combination of the excitations of two nerve centres, by means of their simultaneous excitation of a third? We know of the extension of afferent irritations from one nerve centre to another, and the consequent excitation of motor centres, and the overflow of this along centrifugal tracts. We know of the influence of the auditory word centre upon the Broca centre, as a special case of sensory-motor excitation. We know of the extension of irritations from one sensory centre to another, with consequent shifting of peripherically referred sensation; and we know of the inhibition of motor centres, visceral, vascular, or muscular, by the excitation of non-motor centres. I think this exhausts the known precedents for theories on the interaction of nerve centres. None of them in the least resembles the case imagined by Flechsig. It seems to me that his third postulate must be rejected on the ground that, for the mystery which enshrouds brain processes, it has substituted pure nonsense.

There is no such thing as a complex sight-touch impression. We are not, therefore, concerned to show how such an impression, of which we have no knowledge whatever, might possibly be formed. What plainly happens is that an object presented simultaneously or successively before the retina and an organ of touch, as the hand, excites visual and tactile impressions, and these unite *in*

consciousness to constitute a complex experience of the object. There is not the slightest indication that they unite anywhere else. Flechsig's elaborate argument would never have been constructed, but for the desire to demonstrate the working of cerebral mechanisms entirely irrespective of consciousness. The union of two impressions in consciousness is in itself a somewhat metaphorical way of saying that the mind is, at the same time, conscious of two aspects of the same object.

There has been no combination in space, and such a combination, which has never been proved, is impossible to definitely imagine. The combination has been affected between two events in time.

The gist of the entire question lies in this most simple and elementary case. It is not worth while, therefore, at this time to follow the same line of thought into consideration of the numerous mental acts which are remote from sense perceptions. From the elementary case we infer that the excitation of a sensory centre becomes the occasion for diffused excitation of all other parts of the cortex, except, possibly, other sensory centres. There is nothing to indicate that the secondary excitation is limited to any special locality; hence nothing to indicate that for mental purposes any more significance attaches to the adjacency of cortical centres than to the precise localization of the first or second violins in an orchestra, where also, separated masses of molecular movements so focus into unity as to produce musical sounds. The unifying focus is the auditory consciousness of the listener, without which, as has been said, a symphony by Beethoven would remain nothing but the scraping of horses tails over cats' bowels. Consciousness is the unifying focus for all such processes in the brain as cross its threshold. Subliminal processes are not unified, and thus are neither sensory nor psychic.

The discovery of the memory residues of specific sensory excitations in non-sensory regions adjacent to the sensory centres, would, were it possible, tend to exhibit

some such interaction between the two as Flechsig's theory postulates. But this discovery has not been made. Munk's experiments on so-called psychic blindness and deafness only show the effect of extirpating the entire cortical expansion of the optic or auditory nerves. The animal then, naturally, became unable to see or hear, but it was impossible to show that he, still less a higher animal, had, therefore, lost all previously acquired experiences of vision or hearing. Certainly this is not the case with adult human beings who have become cortically blind. Flechsig admits that the sensory residues of sensory impressions are not stored up in the sensory centres themselves.

If for Flechsig's obscure and clumsy theory we substitute in part Meynert's suggestion, that whenever any event occurs at any level of the cerebrospinal system, the nervous perturbation it occasions is propagated to the cortex of the brain, whose elements retain an organic memory of the excitations they have experienced—we remain within the limits of simple and unbiased observation. Guided by this suggestion, we may, indeed, occasionally detect an influence of adjacency, which otherwise would be inexplicable.

Thus, as I have mentioned, Flechsig is compelled to abandon his combination theory when called upon to explain by it the relations of the frontal lobes to the olfactory sphere. He tries to show that in regard to this, human development does not follow, but contrasts with the general order of phylogenetic evolution; because "in man the sense of smell has become relatively insignificant, and its encephalic organs relatively atrophied; and correspondingly medullization, that is function, begins with the important sense of touch, and not with the unimportant sense of smell."

Flechsig admits, however, that medullated fibres are formed in the olfactory sphere at about nine months, that is, at the same moment that the pyramidal tract and also

many sensory tracts are ripening. Further, the greater importance of the sense of touch in later life does not contravene the earlier importance of the sense of smell at birth, by which even the human young seem to be chiefly guided in fumbling for the breast. Lower down in the animal scale, and among the earliest vertebrates, the sense of smell probably remains throughout adult life the chief guide in the search after food, thus of prime vital importance. In this fact may perhaps lie the reason for the subsequent development of the brain mantle around the originally enormous olfactory organs of the vertebrates. The primal instinctive activities are guided by these organs. When these activities begin to be recorded, so as to construct a continuous experience, a brain cortex begins to be formed and continues to develop in proportion to the increasing complexity of the record. As the sense of personality is undoubtedly associated with the development of continuous experience, and of the record by which the continuity is maintained, it would be approximately and historically correct to say that the sentiment of conscious personality had originated in acts of smelling. This, however, is quite different from saying that the sentiment of the *ego* had been mechanically generated by excitations of the organs of smell. The one plausible basis for Flechsig's speculations is the indisputable fact that masses of gray matter are, in the human brain, intercalated between the cortical areas which constitute the central expansions of nerve tracts directly exposed to external impressions. Further, that within the human species, the range of mental existence seems to be approximately correlated with the amount of gray matter which is so intercalated. Direct examination of the structure of these superadded convolutions, discovers in them mechanisms which would seem to provide for the infinitely multiplied propagation of neural impulses or excitations, and also for the impact of these upon ganglionic cells in a manner to secure something for which the electro-chemical science of the day

supplies the metaphor of storage. As a working hypothesis, and as a substitute for the earlier metaphor of the daguerreotype sensitive plate, we imagine a series of chemical changes, of molecular movements effected in the protoplasm of the ganglionic cells, or of their prolongations, which movements, having once occurred, may be revived by a recurrence of the original excitations, or of secondary excitations which traverse the same lines as the first.

According to Meynert, the brain cortex records all the events, of whatever nature, which transpire within the sphere of existence of the individual. The record is voluminous in proportion to the complexity of the experience acquired. The brain cortex of the new-born infant represents inherited ancestral experience, both phylogenetic and personal. The existence of the record at this time shows that such experience is separable from the brain, because transmissible by the infinitesimal speck of matter which constitutes the germ cell. The more voluminous the inherited mass of cortex, the greater the facilities afforded for recording the events of the new life which are about to transpire. Among the millions of cortical cells we are almost compelled to imagine infinitely complex series of secondary excitations duplicating, reduplicating, endlessly refining upon the original excitation. So that, even when this has arisen in a sensory centre, the sensory impression drops out of consciousness, and only its remote memory, shadow, symbol remains, based upon the series of mental events it was suggested or started. As the complication of the secondary excitations depends upon the number of nerve elements which can be excited, and this number is an inheritance from antecedent circumstance we may say in a general and metaphorical sense, that the new experience of the individual blends with the recorded experience of his family and race, to constitute the total experience of consciousness at any given moment. No space or material combination can be imagined, but a blending in time of an in-

finite series of events. The facts of specific quantitative inheritance show that during the process of making new records, that is during the individual life of the brain, its organic memories, its inherited habits are revived, and these modify the manner of the new recording. The records are not uniform to the same excitation, but assume specific forms which vary in each individual case. There is no moment in which external impression is not followed by specific reaction.

In all this there is no evidence to be found for the localization of psychic functions or faculties, but rather for the totality of brain action at any given moment to any given excitation.

I think the flimsiness of Flechsig's argumentation depends on the futility of his attempt to demonstrate that the brain is the mirror of mind rather than the expression of mind. The attempt has often been made before, but it is interesting to note how each in succession fails. And to this last and brilliant effort in the same direction it has seemed to me eminently worthy to invite the thoughtful criticism of this society.

Note.— Criticisms upon Flechsig's conclusions, and their cases, have not been lacking from other points of view than those developed in the preceding considerations.

Dejerine (*Soc. de Biol.*, Feb. 20, 1897) protests vehemently against Flechsig's view that "only a third of the cerebral cortex is provided with projection fibres, the other two-thirds serving only to associate together the sensorial spheres, and the sensory-motor sphere." "This view rests upon the study of the brains of new-born children, or of those younger than five months: it is in absolute contradiction with the teachings of normal anatomy and the study of secondary degeneration." "All the cerebral cortex contains projection fibres, including, probably, the insula." "It is very possible that the infantile brains studied by Flechsig were deprived of projection

fibres. But it is inadmissible to say that *therefore* these did not later exist." "It is not astonishing that the sensory and sensori-motor centres develop earlier than other regions of the cortex, for they belong to an earlier order of phylogenesis."

CONCERNING THE STRUCTURE OF THE SPINAL GANGLION CELLS OF MAN. (Archiv für Psychiatrie, Band 29, Heft 2.) By M. v. Lenhossék.

Lenhossék wisely says it is necessary that we should be familiar with the normal structure of the spinal ganglia if we hope to be able to locate the commencement of tabes in these parts, and he is able from his studies on ganglia from an executed man to greatly further our knowledge of these structures. He has used bichlorid of mercury for hardening, and recommends toluidin blue as superior to methylene blue or thionin. The iron hematoxylin (Heidenhein) stain is also useful. Eosin or erythrosin may be employed as an after-stain.

Some of the cells of the spinal ganglia are even larger than the giant cells. Lenhossék now regards the space, so frequently seen after hardening, between the spinal ganglion cell and its capsule, as an artifact. In cells properly fixed there is no so-called pericellular lymph space in the spinal ganglia. He thinks with Flemming that fibrils are present in the axis cylinder, and are especially distinct at the portion where this passes into its conical enlargement. He has observed that these fibrils assume a spiral arrangement in passing into this cone. The axis cylinder differs from the cell body in the absence of chromophilic elements, in the presence of fine fibrils, and in its staining and refractive properties. A true cone of origin seems to be present only in the largest cells. The tigroid substance (a name suggested by Lenhossék for the chromophilic elements) in the cells of the spinal ganglia is granular. These granules are especially numerous along the outer part of this substance, but in the larger cells they are not found in the zone about the nucleus, nor in the cone of the axis cylinder, nor in the peripheral zone of the cell. The width of the peripheral zone varies greatly in different cells, and Lenhossék thinks it may possibly become smaller during the activity of the cell. He employs the term chromophilia for a condition depending on the density of the ground substance, and having nothing to do with the tigroid granules. He does not believe these granules are precipitates, for he has found them in the cells of the dog examined immediately after death, without the addition of any substance. Other arguments are also brought forward in favor of this view. Lenhossék has not been able to observe the fibrils in the ground substance of the cell which Flemming has described, on the contrary, this substance seems to him to consist of a reticulum of fine granules. The nucleus of the spinal ganglion cell in man has a membrane and a large solitary nucleolus, which is usually located at the centre of the cell. This nucleolus is usually homogeneous, though occasionally vacuoles are present, and, in the dog, at least, granules may be seen. The nucleus has little structure and is acidophilic. The nucleolus is basophilic, but only to a limited degree.

SPILLER.

A FORM OF TOTAL THIRD NERVE PARALYSIS, TYPICAL OF A UNILATERAL NUCLEAR LESION; WITH A CASE.

By C. A. WISHART, M.D., Pittsburgh, Penna.

Ophthalmologist to the Eye and Ear Hospital of Pittsburgh; Alternating Ophthalmic Surgeon to the Pittsburgh Free Dispensary.

Mrs. B—, a strong and healthy-looking white woman, thirty years of age, was first seen at my clinic at the Eye and Ear Hospital, August 11th, 1897. She stated that on July 21st last she had had an attack of vertigo, of moderate severity, which passed off completely in the course of a day or two, and one week later she noticed drooping of the lid of her left eye. She said that she had always been in good health, had never had a miscarriage and positively denied any specific taint.

Examination.—In the R. E. the pupil measures 3 m.m. in diameter and reacts promptly to light and accommodation. Vision 20/20, with plus 3 sph. 20/20. With correction (plus 3.D) reads Jaeger No. 1, 8—21 ins. In L. E., the pupil measures a little over 6 m.m. in diameter and fails to contract to light or accommodation. Vision 20/200, with plus 3 sph. 20/20 nearly. In this eye (the left) there is complete paralysis of accommodation, complete ptosis, divergent strabismus and loss of movement in all directions except outward. There is secondary deviation of the right eye outward. Crossed diplopia, the image of the left eye being higher than its fellow and upright, the upper extremity of the image of the right eye being inclined to the right. The lateral distance between the images increases as the test-object is carried to the right. If the test-object is moved upward, the difference in height and the inclination of the image of the right eye increase.

The loss of movement, inward, upward and downward, of the left eye, indicates paralysis of the internal, superior and inferior recti, while the preservation of outward movement and the upright character of the image, shows that the external rectus, superior and inferior obliques, are not affected. There is no apparent limitation of movement in the right eye, but the upper extremity of the image seen by this eye is inclined to the right. In paralysis of an inferior oblique muscle, the upper extremity of the false image is inclined toward the affected side, i. e., in paralysis of the right inferior oblique the upper extremity of the image in the right eye is inclined to the right side.

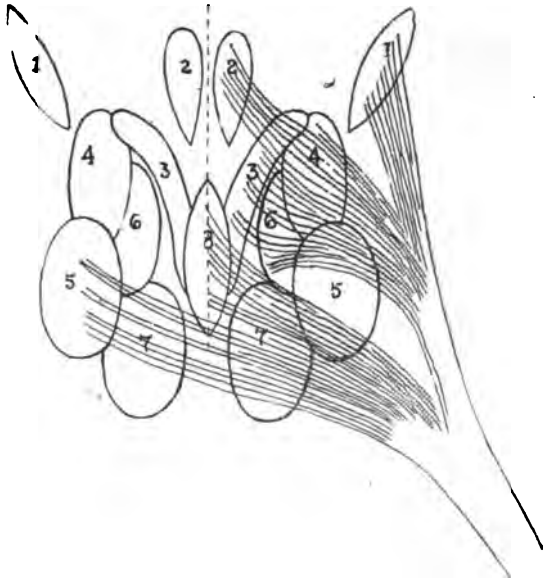
There is, then, in this case paralysis of all the muscles in the left eye supplied by the third nerve except the inferior oblique, and in the right eye paralysis of the inferior oblique alone, i. e., paralysis of one complete set of muscles supplied by the third nerve, but distributed in a particular way between the two eyes.

We are all familiar with the description of total third nerve paralysis given in the books, which is substantially as follows: There is crossed diplopia, the image of the affected eye being higher than its fellow, and its upper extremity inclined to the affected side the lateral distance between them increasing as the test-object is moved toward the sound side. If the test-object is moved upward, the difference in height and the inclination of the false image increase. There is divergent strabismus and limitation of movement in all directions, except outward and slightly downward. The secondary deviation of the sound eye is outward, the false projection of the field of vision is to the inner side, and the face is inclined toward the affected side, the chin being tipped upward. In addition, there is ptosis, medium dilatation of the pupil, which fails to contract to light, and paralysis of accommodation.

All of the authorities that I have been able to consult adhere more or less closely to the above description as typical of total third nerve paralysis, and endeavor to differen-

tiate between cases due to a peripheral lesion and cases due to a nuclear lesion. In my opinion, this description is applicable only to cases of total third nerve paralysis due to a peripheral lesion, while the case which I report above is typical of total third nerve paralysis due to a unilateral nuclear lesion.

In support of this opinion, it will only be necessary to



Schematic Diagram of the Nuclei of the Third Nerve, showing Decussation of Fibres to Inf. Oblique. (After Knies.)

1. Sphincter Iridis; 2. Levator Palpeb.; 3. Ciliary Muscle; 4. Sup. Rect.; 5. Inf. Oblique; 6. Int. Rect. (conjugate movements); 7. Inf. Rect.; 8. Int. Rect. (convergence).

call attention to the manner in which the root fibres leave the nuclei.

Jakob¹ says: "The nerve fibres pass out of the nucleus as the roots of the motor oculi on the same side, in

¹Ch. Jacob, *Atlas of the Normal and Pathological Nervous Systems*. Wm. Wood & Co., 1896.

lesser part also decussating with the fibres of the other side, and run in the motor oculi trunk to the muscles of the eye." Hill² says: "Most of the fibres come from the gray matter on the same side of the brain, but some cross to the opposite side before taking exit." Knies³ says: "Hence the motor oculi nucleus of each side contains the nuclei of those muscles which take part in the movement of both eyes toward the opposite side, i. e., the internal, superior and inferior recti of the same eye and the inferior oblique of the opposite eye." Granting the statements of these authorities to be correct, the accompanying figure, modified from Knies, will illustrate how a unilateral lesion affecting the whole of the third nerve nucleus on the left side of the brain, must result in the particular form of paralysis found in my case.

Therefore, to Starr's conclusion⁴ that "if all the muscles of the eyeball supplied by the third nerve are affected, including the iris, the case is one of total peripheral paralysis of the third nerve, and the lesion lies on the base of the brain," I would add the following, "but if all the muscles of one eye supplied by the third nerve are affected, except the inferior oblique, with paralysis of the inferior oblique alone of the opposite eye, the case is one of total unilateral nuclear paralysis of the third nerve, and the lesion lies on the same side as the eye in which the inferior oblique is not affected and on the opposite side to the eye in which the inferior oblique is alone affected."

²Alex. Hill, Article in Norris & Oliver's System of Diseases of the Eye. Vol. I.

³Max Knies, The Eye in General Diseases. Wm. Wood & Co., 1895.

⁴M. Allen Starr, Journal of Nervous and Mental Disease, May, 1888.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, October 5, 1897.

B. Sachs, M.D., President.

A DISCUSSION OF THE PARESTHETIC NEUROSIS:—PSYCHROESTHESIA AND KAUMAESTHESIA.

Dr. C. L. Dana presented a paper with this title. He said that paresthesia included nearly all the subjective sensations of the skin except those of pain. When these sensations fastened themselves to a particular part, as a nerve, they developed a definite picture, and were as much entitled to a distinctive name as was neuralgia. Sometimes paresthesias of the head caused sensations of burning, pressure and cold, which were entirely comparable to headache. They affected the cerebrospinal nerves just as did neuralgias. The cephalic paresthesias were usually symptomatic of a lithaemic state. The most frequent causes of local paresthesias were those concerned with occupations. Women were affected more frequently than men. The feet and legs were most affected; next the hands alone, and next the hands and feet together. The nerves most affected were the brachial and their branches. The most common form of paresthesia was a sensation of tingling or numbness; more rarely there was a sensation of heat. Among the rarest forms of paresthesia were sensations of cold, which were entirely apart from an actual lowering of the temperature of the part, and which occurred without any objective vascular changes. This form was not usually very distressing. The term "psychroesthesia" was first applied by a French physician in 1886. The reader of the paper said that he had himself met with a number of these cases, of which the following were illustrations:

Case I. Dora C., a washerwoman, had suffered from chronic tinnitus for three years, and had some disease of both middle ears. She complained especially of a cold sensation which she had felt continually in the forehead

for three years. The sensation was bilateral, and involved the upper part of the forehead. The skin was not cold to the touch, nor did it appear in any way abnormal. Examination revealed no anesthetics, and no signs of organic disease.

Case II. A man, fifty-six years of age, a mechanic. He was compelled to stand all day at his work. For a year and a half he had some paresthesia of the lower part of the legs, and had also suffered from a distressing sensation of cold in the foot. The physical examination showed absolutely no anesthesia of the affected part, and no change in vascularity. The reflexes were slightly exaggerated.

Case III. A man, forty-two years of age, a butcher, whose previous history was negative. He complained of a sensation of cold over the left thigh, particularly its anterior surface. This sensation had been continuous for the past six months, and was increasing in severity. There were absolutely no objective signs over the affected part. The man was a dyspeptic.

None of the cases, the speaker said, was an example of beginning or terminal alcoholic neuritis, in which paresthesias are so common. He had noted particularly two classes of cold anesthetics, viz., one, not definitely limited to certain areas, but involving the whole extremity or all four extremities; and the other, to which the name psychroesthesia proper should be given. The former was associated with pain or vasomotor disturbance, and was due to irritation of the peripheral nerves; it indicated an abortive type of degenerative neuritis. Cold sensations were very rare when neuritis was marked, as in alcoholic neuritis. Such diffuse cold sensations also occurred in syringomyelia, and in lesions of central gray matter of the spinal cord. The second class represented a disease in which the patient suffered from a sensation of cold exclusively, there being no associated tinkling or prickling. It was usually confined to some small area. These patients felt as though some cold object were lying upon the part. These sensations were usually of traumatic origin, and were associated with lithemia, and with the degenerative changes of middle life. In his experience, they had occurred more often among men than women. Apparently, the cold paresthesias were not produced by lesions of any of the sensory neurons, that is, by any lesions above the

spinal ganglia. The only two conditions in which cold sensations were found were: (1) Lesions of the central gray matter, possibly involving the terminals of the first sensory neuron or the beginning of the second sensory neuron (e. g., in beginning syringomyelia); and (2) in lesions of the very terminal portions of the peripheral filaments. The pure types of psychroesthesia he considered to be always due to irritation of the peripheral filaments, and the cold paresthesia found in tabes and various lesions of the spinal cord were always mixed pains.

Dr. William H. Thomson said that he had a case to report which was not in harmony with Dr. Dana's decision with reference to the central relations of psychroesthesia. On March 7, a gentleman, fifty-one years of age, had called upon him, stating that he had awakened the night before with a sensation of "universal numbness" over his right side. It involved the face, right arm and legs, and the numbness was accompanied by a prickling sensation, and by a binding sensation just above the right knee. The pulse was 94 and of a high tension; the artery was somewhat thickened; there was no real anesthesia to pain. There was a decided increase in the knee reflex on the right side. There was no aphasia or deviation of the tongue. His special complaint was a sense of coldness distributed over the shoulder and down the arm, involving the fingers and back of the hand, but more particularly the leg and foot. The urine showed no albumen or casts. The case was kept under observation until June 4. At that time it was noted that there was the same distribution of the cold sensation, except in the face. There was also a pronounced redness of the skin extending from the crest of the ilium to the knee, but quite as marked on the other extremity as on the affected side. The speaker said that the lateral distribution, involving the face, seemed to be an exception to the cases reported in the paper.

Dr. George W. Jacoby thought Dr. Dana was right in ascribing many of the one-sided paresthesias to neuritic conditions, but he was of the opinion that the symmetrical paresthesias were due to some general condition—one which exerted an influence on the central gray matter of the spinal cord. This general condition was usually a toxemia of some kind—quite commonly an autointoxication from the intestines. He had seen a number of examples of paresthesias resulting from the inordinate use of tobacco. In these cases there was a general feeling of heat extending down one or both arms, along the distribution of the ulnar, and such a paresthesia was to him almost symptomatic of tobacco poisoning as the

etiological factor. Another characteristic paresthesia was a sensation of heat or tingling passing along the penis and into the scrotum; and a third was a paresthetic condition distributed along the inner part of the thigh, usually symmetrically. These three paresthesias he had found very frequently in persons using tobacco to excess, and they had disappeared after the use of the tobacco had been *entirely* given up.

Dr. C. A. Herter said that he had met with several instances of paresthesia of cold in which the distribution was somewhat different from that mentioned in the paper. For example, he had twice met with paresthesia on the abdomen, and also upon the chin. He agreed with the reader of the paper in ascribing most of these cases to peripheral irritation of the nerves, but whether this irritation depended upon auto-intoxications was a matter about which we could not speak very certainly as yet. In one of his cases there had been an actual lowering of the surface temperature of about one degree, as compared with the other side. He could not say whether or not this was a common feature.

Dr. William Hirsch said that these cases were in all probability due to peripheral lesion, or were cases which had developed as a result of chronic alcoholism. He had seen four cases (including one he had already presented to the Society) in which trauma had acted as an exciting agent. One patient, while traveling, had carried another passenger in his lap for a long time. Following this he had developed a paresthesia of the thigh. Another patient, also a sufferer from chronic alcoholism, had experienced trauma in the upper portion of his thigh. More than one of them had noticed, on taking a hot bath, that the part complained of did not feel heat with the normal acuteness.

Dr. W. M. Leszynsky said that he had seen three cases, all in persons nearly fifty years of age. One was a man with general atheroma, who had a large area of cold sensation over the lower part of the leg. He was given nitroglycerine, and the sensation disappeared. In another case, the cold feeling existed for a long time, and then was succeeded by a sensation of heat. Subsequently the hot and cold sensations alternated. In still another case, the urine was of high specific gravity, and contained indican in excess. One of these last two cases improved decidedly.

Dr. A. D. Rockwell asked if Dr. Dana included among his cases those paresthesias which result from acute diseases—e. g., typhoid fever—for, he could recall a great many instances of paresthesia following such acute diseases.

Dr. Frankel said that he had examined the sensory disturbances in tabetics, paying particular attention to temperature

paresthesia. Out of 30 tabetics, only 2 had cold paresthesia. One complained constantly of a cold sensation along the back and down the legs; the other complained of cold paresthesias along the back part of the legs and the extensor surfaces of the upper extremities. The general appearance of this case was rather that of cerebrospinal syphilis, and the condition appeared to be the result of some meningitic pressure. There was no disturbance of the temperature sense in these cases. The last case he would explain by pressure on the posterior horns simply.

Dr. Leopold Stieglitz had seen some paresthesias of the temperature sense—one very marked one in a case of multiple sclerosis. The patient had been under his observation for five or six years, and had suddenly begun to complain of a sensation of cold extending from the umbilicus down through both legs, as if he were standing in cold water up to the waist. This sensation lasted about two weeks, and then gradually wore off. Following this were occasional sensations of cold in one or both extremities. Probably in this case it was the result of transient disturbances in the circulation in the spinal cord. The examination of the patient at first showed a diminution of sense of cold, much more than of heat. He did not think all the cases were due to peripheral neuritis; in some the paresthesia seemed to be an early symptom of tabes.

Dr. J. F. Terriberry said that inasmuch as tobacco was supposed to play such a small part in the peripheral sense apparatus, outside of the optic nerve, he had been deeply interested in the remarks of Dr. Jacoby. It was well known that under certain circumstances tobacco was a decided depressant, and an excitor of dyspepsia, and it was possible that it acted indirectly in this way by causing autointoxication.

The President said that he had seen a number of the special forms of pure paresthesias described in the paper. One case was that of a physician, thirty-five years of age, who had been moderate in his habits, yet ever since his student days had had a sensation of cold on the inner aspect of the left thigh. This was aggravated by severe exercise, but not influenced by changes in the weather. The examination was entirely negative, and treatment had no effect. The speaker said that he had been particularly troubled with a class of cases with a persistent burning sensation in the heel. This was not affected by exercise or by posture, or by treatment, either surgical or medical. He thought these cases were generally due to some lithæmic or gouty condition; he had suspected ostitis or periostitis in some cases.

Dr. Dana, in closing the discussion, said that the more pure the cold paresthesia, the surer might one be that it was

a lesion of the peripheral nerves and terminal filaments. In some of his cases, similar to that described by the last speaker, there had been originally an eczema, or some slight form of trauma, which had ultimately become a mere dermal illusion. Regarding the case reported by Dr. Thomson, he said that it compelled him to admit that those paresthesias might be caused by central brain lesions. He could confirm Dr. Jacoby's statements regarding the effect of tobacco, for he had seen obstinate and distressing paresthesias of the hand, which disappeared very rapidly on giving up the tobacco. The authors that he had consulted had not made systematic observations of the temperature of the skin. In reply to Dr. Rockwell he would say that after pneumonia and typhoid fever he had only seen the general anesthetics such as were observed in the mild types of neuritis. He had had some experience with the troublesome cases of "burning heels," and had come to believe that many of them, at least, were due to a certain degree of traumatism, resulting, perhaps, in periostitis.

A CONSIDERATION OF FLECHSIG'S "GEHIRN UND SEELE."

Dr. Mary Putnam Jacobi read a paper with this title. (See page 747.)

Mr. Marshall said that psychologists looked with some doubt upon the positive statements made by many of the modern neurologists. It seemed rather startling that the neurologist paid little attention to psychology. He was certain that if Flechsig had followed the development of modern psychology, he could never have written the book under consideration. He agreed most heartily with all the criticism presented by Dr. Jacobi. It was impossible to hold any such view of sensation as Flechsig apparently did. It could not be looked upon as though existing alone—it was a part of the whole pulse of consciousness—a sort of "streak" in our conscious life. The whole mass of the consciousness of the moment must be taken into account. Sensation seemed like the top of a wave, or a particular kind of increment to this mass of consciousness. Flechsig's position certainly seemed to be determined by the old-fashioned view of the separation of the soul from the mind, but modern psychology considered consciousness only—the soul, the *ego*, was a part of consciousness. Consciousness from a certain standpoint could be looked upon as divided into the field of attention and the field of inattention. There was no absolute separation between the two. Stout, a

recent author, distinguished between moetic and amoetic consciousness, and the soul is a part, if not the whole, of this field of inattention. His own personal view is that the "empirical ego" was nothing more than the field of inattention to which the items of attention are added. Flechsig's view of association also was not in accord with modern doctrine, and the whole notion of association, as held in the early part of the century, was fast disappearing. The notion of the addition or summation or semi-chemical combination of sensations had been entirely dropped. Psychologists also looked with some doubt upon the statement of the neurologists regarding a record in the cortex, for nothing in the consciousness gave any reason for believing this. The psychologist believed that it was impossible for any two successive stages of conscious life to be the same. Each addition to our psychic life changed the whole of it, and there was no such thing as the reduplication of any conscious phenomenon, so that the notion of "storage" and physical residue or record in certain centres was not acceptable to the psychologist. Each added state changed the whole pulse of mental life; apparently the brain itself was altered as a whole.

Dr. S. E. Jelliffe remarked that he thought that Flechsig's idea broke down when he explained the intercalation of the frontal lobe between the olfactory and somesthetic areas.

Dr. Hirsch said that the attacks on Flechsig's theory had become exceedingly numerous. To his mind, the principal theory did not seem at all new; it simply consisted in dividing the psychical functions of the brain as such from the lower functions, i. e., the different sense perceptions and the lower motor functions. The really new thing introduced by Flechsig's theory was the localization of these two functions in special areas of the brain. His reasons for such localization were: (1) The later development (as Flechsig claimed to have proved); and (2) that these purely psychical areas belong exclusively to the association system. If this could be proved, the whole theory would stand. The objections of H. Sachs, of Breslau, were based on some published pathological cases. Among others, there was a case in which the whole occipital lobe and the part of the cortex which Flechsig claimed to be the entire perception centre for vision, were destroyed. According to this theory, there should have been a degeneration of the optic coronary system, yet H. Sachs found this system intact, and he concluded, therefore, that this system must necessarily be in connection with other portions of the brain, and hence, that Flechsig had no right to claim that the parietal convolutions were association centres. The speaker said that he could not accept this argument of H. Sachs. Any degener-

ation in this optic coronary system must be centripetal; hence H. Sachs had no right to expect a centrifugal degeneration.

With regard to the differences of development, the speaker said that every other organ of the body was perfectly developed as regards function immediately at birth—e. g., in the case of the heart or the kidneys—yet the brain of the new-born child was far different from that of an adult. Why should not this well-known gradual development of the brain have the anatomical basis assured by Flechsig? He thought, with Flechsig, that there was a certain proportion between the time of medullization and the development of function. From a purely psychical point of view it seemed to him that Flechsig's theory could be sustained. The idea of the different localizations of the intellectual powers was in full accord with our psychiatric experience. One portion, it was well known, could be diseased without intellectual impairment. This was seen every day in cases of melancholia. The anatomical part of Flechsig's theory was undoubtedly open to much criticism, but Flechsig had opened a new channel of investigation—a combination of anatomical and clinical research—which should make his theory welcome as an attempt which would, in time, bring great benefit to psychology.

Dr. Joseph Collins said that, if time permitted, he could show that the reader of the paper had not done Flechsig justice, at least from an historical standpoint. For instance, statements made more than twenty years ago should not be cited as reflecting that writer's state of mind or belief to-day, especially when such statements are at variance with recent utterances. The essay, "*Gehirn und Seele*," had been followed by a torrent of criticism, but many of the objections had been satisfactorily controverted in the author's last essay on cerebral localization. When this last essay was read at the Congress of Psychologists at Zurich, psychologists in both Europe and America were very much in accord in the admission that it satisfied their faculties of ratiocination at that time, and Baldwin, of Princeton University, in his report of the paper to an American periodical, had no criticism to make, except to say that the claims should not be accepted too hurriedly. Flechsig's anatomical contentions have been cited in great detail by Van Gehuchten in the recent edition of his admirable work, and, although, he does not say in so many words, that he is partisan to all of Flechsig's claims, the space given to these views in his book may be taken as an index of their importance in the author's estimation. Other reputable anatomists and psychologists have corroborated many of Flechsig's claims, and it would, therefore, seem to the speaker that the necessity

of proving the attributed speciousness of these claims was enormously with his critics.

It could be shown that the localization of functions in certain areas of the brain, as stated by Flechsig, coincided exactly with the allocation of function to these parts by clinicians and psychologists the world over. The central visual representation was in the occipital lobe, and especially in that portion known as the cuneus, an area to which it was confined years before by Seguin, Henschen, Wilbrand, et al. The "Körpergefühl" area of Flechsig corresponded with the area in the central convolutions which Dana had shown from clinical and physiological experience was concerned in the interpretation of tactile sensibilities, and so on throughout the entire list of centres. The same might be said of the association areas which Flechsig believes are intercalated between areas of specialized or highly specialized functions.

Mr. Marshall had said that the notion of "storage" and of physical residua or record, the result of impingement of sensory stimuli on the brain was not acceptable to the psychologist, and the speaker was glad to hear that they were as sensitive to the imperfections of this nomenclature as was the physician, for, except as a colloquial expression, it was not acceptable to the latter. Yet, until a word can be found which will explain that the potentialities of a specialized area of the brain is changed by having given reception to a stimulus, whether the change be a chemical, a psycho-physical, or a physical one, we shall have to make use of this term at its current value, which is moderately stable, and not in a literal sense. Probably no one was ready to accept Flechsig's teachings in toto, but they are too important, and too uniformly in accord with the teachings of anatomy, physiology and psychology, to be dismissed after a few minutes of scholastic indulgence or with a personal estimate of flimsiness.

The President said that he had discussed this subject in an address delivered at Baltimore last May. He had always had unbounded confidence in anything published by Flechsig, and had gone at the task very seriously; yet he did not think that anyone who had had any sort of training in psychology or philosophy or logic could accept Flechsig's presentation of the subject. First of all, the evidence presented was insufficient. This deficiency had been acknowledged quite recently even by one of Flechsig's ablest students. The theory of the coincidence of function with the development of the various medullated tracts seemed to have been carried too far. If Flechsig's theory were correct in this point, why should speech fibres be developed fully (as he said) at the third or fourth month of life? We were all agreed as to the extent of the

somesthetic area, but his argument went further. Flechsig had taken almost the entire outlying districts and called them association *centres*, intellectual centres, not tracts. These centres, he said, simply connected the various areas of the brain, and connected sensory with motor areas. If true, the frontal lobe's chief function should be to combine the somesthetic area with the sensory speech area or tactile area. From our knowledge of tumors in the frontal lobe it was evident that there were other very important functions than could be assigned to it on the basis of Flechsig's theory. The latter seemed to him to be open to a number of serious objections which he had stated in some detail in the *American Journal of Insanity*.

Dr. Jacobi, in closing the discussion, said that no one could doubt the position of Flechsig as an anatomist. It was not necessary to dispute what he had established anatomically; her object had been to show how much that was not anatomical he had added to pure anatomical facts. There was really very little foundation of fact for all his fanciful conclusions.

THE MANNER IN WHICH THE LAW FORBIDDING THE PUBLIC REPRESENTATIONS OF HYPNOTISM OUGHT TO BE MODIFIED. By Dr. Crocq., Jr. (*Journal de Neurologie et d'Hypnologie*, No. 15.)

C., in discussing the subject under title, arrives at the following conclusions:

1. Hypnotism presents great dangers for the subjects (*sujets*) and for society.
2. It is absolutely necessary that the law should prevent the occurrence of these accidents.
3. It is possible to simulate all the experiments of hypnotism.
4. In educating (training) an individual one can succeed in developing in him a state which has all the appearances of the wake state, and yet reduce the individual to a condition of complete automatism.
5. It is sometimes absolutely impossible for the most experienced expert to say whether or not an individual is under hypnotic influence.
6. The public representations of simulated hypnotism present absolutely the same dangers as do the public séances of actual hypnotism.
7. In order to entirely prevent these accidents, the law must forbid not only the real representations of hypnotism but also the simulated ones.

Drs. Delbœuf, De Rode and Crocq., Jr., were asked as experts to investigate whether Donato's public demonstrations of hypnotism were indeed simulated, as he swore them to be before the courts of Belgium, or real. The said experts made Donato repeat before them the experiments which he had demonstrated before the public; they further repeated these experiments themselves and found that the "subjects" (*sujets*) were perfectly awake during the whole time of the experiments.

ONUF.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. HENRY L. SHIVELY, M.D., N. Y.
WM. C. KRAUSS, M.D., Buffalo, N.Y. A. STERNE, M.D., Indianapolis.
W. M. LESZYNSKY, M.D., New York

NEUROPATHOLOGY.

FOCI OF DEGENERATION IN THE WHITE MATTER OF THE SPINAL CORD IN LEUKEMIA. (*Deutsche Zeitschrift für Nervenheilkunde*, Band X., Heft 3 u. 4.) By M. Nonne.

Comparatively few examinations of the central nervous system in cases of leukemia have been published. Nonne reports the changes which he has found in the spinal cord in two cases of this disease. These consisted of unsymmetrical foci of various sizes in the white matter. The axis cylinders in these foci were swollen; empty spaces were found, and the neuroglia was proliferated. No round cell infiltration, and no alterations of the vessels were noted. The columns of Goll, in the first case, were somewhat sclerotic throughout the cervical, and a part of the thoracic region. The gray matter and spinal roots were normal. No connection of the foci with the vessels could be observed. The lesions were very similar to those which have been described in pernicious anemia. Nonne calls attention to the fact that the foci may be very minute. SPILLER.

MYELOPATHIA ENDOARTERITICA ACUTA, WITH OBSERVATIONS ON "PRESSURE ANESTHESIA." (*Deutsche Zeitschrift für Nervenheilkunde*, Band X., Heft 3 u. 4.) By E. Biernacki.

Biernacki describes the case of a man of fifty years, who probably had not had syphilis, and in whom was found flaccid paraplegia without pain; loss of the rectal and vesical functions; loss of the skin and tendon reflexes, and decubitus developed within a short time. The symptoms differed from those of acute transverse myelitis in the preservation of tactile sense, although temperature, pressure and pain senses were affected. Fever only developed with the appearance of cystitis and decubitus. No inflammation of the cord could be found, *i. e.*, no round cell infiltration nor granular corpuscles. The changes within the cord were insignificant, and consisted of disseminated foci of swollen axis cylinders. The cells of the spinal cinerea were not normal. Many obliterated vessels were found in the meninges, and the infiltration affected the intima alone. Only the arteries of the posterior spinal system were diseased. The meningeal veins were also involved. The foci within the cord were evidently in relation with the vascular lesions. The paraplegia was thought to be due to the closure of the vessels.

The second case was much like the first. Acute and more chronic, disseminated, inflammatory foci were found within the cord. These evidences of inflammation probably represented a later stage of the same process as seen in the first case.

In the third case, in which chronic hydrocephalus was found, the acute paraparesis and disturbance of the functions of bladder and rectum were ascribed to endarteritis of the meningeal vessels. Bier-nacki lays special weight on the vascular lesions in these cases, and regards all three cases as similar. The lesions within the cord were supposed to be secondary to those of the meningeal vessels, and of more interest pathologically than clinically.

The writer has found that pressure, as for example of the ulnar nerve, causes a dissociation of sensation very similar to that seen in syringomyelia. In typical cases of syringomyelia pressure sense as well as the tactile is preserved, but the former is affected in "pressure anesthesia." In the analgesia of syringomyelia the faradic current does not produce the same sensation of pain as in normal persons, but in the "pressure anesthesia" analgesia to the faradic current is not observed.

SPILLER.

CENTRALE HÆMATOMYELIE DES CONUS MEDULLARIS.—Central Hæmatomyelia of the Conus Medullaris. (*Deutsche Zeitschrift für Nervenheilkunde*, Band IX., Heft 3 u. 4.) By H. Higier.

A young woman, shortly after a fall from a considerable height, presented total paraplegia with loss of sensation, without loss of consciousness. At the same time there was *retentio urinæ et alvi*. After a few days there was not a trace of paraplegia; there was, however, dissociation of sensation in the gluteal and peroneal regions, on the posterior portion of the thighs, and in the mucous membrane of the recto-vesico-genital tract. Retention yielded partly to incontinence of bladder and rectum. A deformity was noticed at the height of the eleventh and twelfth thoracic vertebræ, but there was only temporary evidence of injury to the cord at this portion. The lesion was probably at the lower part of the sacral cord, about the height of the third to the fifth sacral nerves. The patella reflex (second to fourth lumbar nerves) was intact, the Achilles tendon reflex (fifth lumbar, first and second sacral nerves) was lessened, but not abolished. Experiments on animals and post-mortem examinations in man seem to show that the reflex centres for bladder and rectum are located at the level of the third and fourth sacral nerves.

The limitation of the symptoms to the region innervated by the lowest nerves of the cord in a process beginning acutely, and the absence of pain in the affected parts, are points in favor of injury of the conus and not of the cauda equina. On the other hand, there were no fibrillary twitchings, and no gradual extension of the anæsthetic area, which have been mentioned as occurring in conus lesions. A valuable sign of intramedullary affection was the partial paralysis of sensation, as seen in syringomyelia. The diagnosis of hæmatomyelia of the conus was made.

A point worthy of note is the preservation of sensation on pressure in the testicles in lesions of the conus, as these organs are innervated from the lumbar plexus.

The paper presents a careful review of the literature on conus affections, and can only be properly appreciated when read in the original form.

SPILLER.

PSYCHOLOGY.

STATISTICS DEALING WITH HEREDITARY INSANITY. J. Turner (*Journal of Mental Science*, July, 1896).

Turner makes a careful analysis of some 1,039 patients under his control, extending over a period of ten to eleven years, and gives the following conclusions:

I. Direct inheritance. (a) Taking all cases of insanity, acquired and congenital, we find that while the insane father transmits his mental instability to a greater number of offspring than does the insane mother, it is on the daughters that it mostly falls, and where the mother is insane the influence is still more marked in the direction of the daughter, so that, whichever parent is insane, ultimately more daughters inherit insanity than sons. (b) The number of insane mothers is very considerably greater than insane fathers.

II. Reversional and collateral inheritance. (a) In both sexes the stronger influence comes through the maternal branch of the family. (b) The males have the larger number of brothers insane, the females the larger number of sisters. JELLIFFE.

MENTAL STATES ASSOCIATED WITH VISCERAL DISEASE IN THE INSANE.

By Henry Head, M.A., M.D. (*Jour. of Ment. Science*, Jan., 1896.)

The author describes a type of insanity consisting of a quickly changing melancholia with hallucinations and delusions of suspicions. He places more stress on the kind of delusions, the subject, etc., than is usual. In a study of 169 cases of visceral disease, he finds 87 suffering from referred pain associated with superficial tenderness. Sixty of these showed the depression, while of the 82 others none had depression. Moreover he found the intensity of the pain and tenderness to vary directly with the depression. He does not find that local pain and severe diseases produce this depression, but that the depression is associated with areas over the lower part of the chest and over the abdomen. (The study and the paper is described as incomplete.)

MENTAL SYMPTOMS OCCURRING IN BODILY DISEASES. By E. S. Reynolds, M.D., M.R., C.P. (*Jour. of Ment. Science*, Jan., 1896.)

The author finds "pleasurable feelings" to be common in phthisical patients, as also, temporarily, from alcohol, opium, chloroform and cannabis indica. "Mental depression" he finds far more common and accompanying abdominal diseases, rheumatism, influenza, oxaluria, phosphaturia, interparoxysmal states of epilepsy, alcoholic paralysis and movable kidney. "Mental dullness" he finds in various cerebral conditions, in disorders of the liver, in cancer of the stomach, and toward death. "Irritability" he finds in sick children, in adult phthisis and diabetes, in dyspepsia and gout.

Hemiplegics he finds to be usually, and left-sided hemiplegics more probably sane than those of the right side. Aphasia is to be carefully guarded from meaning insanity. He finds mental symptoms rare with chronic spinal diseases. Valvular heart disease he finds to produce insanity, though not in special forms as claimed by Mickle. He has seen two cases of acute mania in the last stages of gouty kidney.

Under diseases caused by germs he thinks there is a larger amount of insanity. Under pneumonia he finds frequent cases, usually of a mild, recoverable form. After influenza and typhoid fever, mania or melancholia may occur. From a large experience in tuberculous cases he regards it as more proven that insanity predisposes to phthisis than phthisis to insanity.

Book Reviews.

LE CERVELET (*The Cerebellum*). An anatomical, clinical and physiological study from the laboratory of Dr. Dejerine. By Dr. André Thomas, formerly Interne in the hospitals of Paris. G. Steinheil, Paris, 1897.

The author has made a very careful study of the cerebellum. We may pass over the chapter devoted to a review of the literature on the physiology of this organ, and direct our attention to the portion which represents the writer's original investigations.

From the study of a case of hemisection of the spinal cord, Thomas concludes: (1) that the cerebellar cortex receives the fibres of the direct cerebellar tract, those of Gowers' tract, and some of those of the posterior columns; (2) that these fibres terminate chiefly in the anterior and superior part of the vermis after decussation; (3) that the fibres of the direct cerebellar tract and of the posterior columns terminate in a higher plane than do those of Gowers' tract. From a case of compression of the spinal cord in Pott's disease with ascending degeneration, studied by the method of Marchi, Thomas concludes that the course of the direct cerebellar tract, and that of the fibres of the posterior columns in the oblongata and restiform body, and that of Gowers' tract in the oblongata and pons, are very similar in man and the lower animals.

Only a small part of the restiform body is composed of fibres from the spinal cord; the greater portion consists of fibres which arise in the oblongata. The restiform body degenerates only slightly from cerebellar lesions. The cerebello-olivary tract contains fibres which arise in the lower olive and terminate in the cerebellar cortex, and not in the dentatum. After a lesion of a cerebellar hemisphere, of long standing, the restiform body and the arciform fibres on the side of the lesion, and the lower olive and the arciform nucleus on the opposite side, may be found atrophied. Thomas destroyed half of the cerebellum in a dog, and killed the animal three and a half months after the operation. He found the cells of Clarke's columns, especially those on the same side as the lesion, atrophied, the cells of the lateral group of the anterior horns in the thoracic region were somewhat decreased in number, and the cells of the lower olive and pontine gray matter of the opposite side had partly disappeared. The reticulated substance of the opposite half of the tegmentum was also atrophied. This experiment showed the rapidity with which retrograde cellular atrophy occurs, especially in short neurones, and the desirability of depending upon the method of Marchi in studying the course of fibres.

Thomas believes that the fibres of the middle cerebellar peduncle arise chiefly in the pontine gray matter of the side opposite to the cerebellar hemisphere in which they terminate, although some may arise on the same side. These fibres end in the cerebellar cortex, and not in the dentatum. A cerebellar hemisphere is connected with one-half of the cerebrum by means of the pyramidal tract and middle cerebellar peduncle, though there is also a connection through the superior peduncle. Atrophy of the middle peduncle from a cerebellar

lesion is partly due to the destruction of certain fibres which arise in the cerebellar cortex, but chiefly to the fact that this peduncle contains short neurones, which rapidly undergo retrograde degeneration.

Thomas has found degeneration of the descending cerebellar tract in the anterolateral column of the cord after cerebellar lesions. These fibres are most numerous in the cervical region, but may be traced into the lumbar. He observed some degenerated fibres in the anterior column of the opposite side. The fibres of the descending cerebellar tract terminate about the cells of the anterior horn of the cord without decussating. They arise in the dentatum, and probably also in Deiters' and Bechterew's nucleus. Some investigators have been unable to observe degeneration of this tract after cerebellar lesions; no one has observed it in man, and Thomas has also been unable to notice degeneration in the lateral zone of the cord as described by Marchi.

Some of the fibres of the superior cerebellar peduncle terminate in the nucleus ruber, but most of them probably pass further forward, though they have not been traced beyond the thalamus. This peduncle probably arises chiefly or entirely in the dentatum. This is at variance with the results of some other investigators, and the explanation for this, as given by Thomas, is that the method of Marchi is much more reliable in studying the course of fibres than any method which depends on the formation of sclerotic tissue. The author speaks of a descending branch of the superior peduncle which terminates, in greater part, in the nucleus reticularis tegmenti pontis.

The principal efferent systems of the cerebellum are the superior peduncle, the cerebello-vestibular fasciculus (internal segment of the restiform body) and the descending cerebellar tract. The principal afferent systems are the restiform body and the middle peduncle.

There are not very many clinical reports in the literature of cerebellar affections with post-mortem findings which enable us to understand the physiology of the organ, for cases of tumor or abscess are not suitable for a study of altered cerebellar functions, inasmuch as many symptoms are produced by pressure. Only cases of incomplete development, absence, atrophy or sclerosis of the cerebellum are useful for a study of the physiology of this organ. Thomas describes a symptom-complex of cerebellar disease formed from suitable cases in the literature. He gives in detail the reports of two cases of cerebellar atrophy observed in Dejerine's clinic, cases which he has studied microscopically. He reports also his results in a large number of experiments on the cerebellum of animals, and finishes this important work with the statement that the cerebellum is a reflex centre of equilibration.

SPILLER.

BOOKS RECEIVED.

"Practical Treatise on Sexual Disorders of Male and Female," by Robert W. Taylor, M.D. Lea Bros. & Co., New York and Philadelphia, 1897.

"A Manual of Medical Jurisprudence," by Alfred Swaine Taylor, M.D., F.R.S., with additions and citations by Clarke Bell, LL.D. Lea Bros. & Co., New York and Philadelphia, 1897.

"A Text-Book of Diseases of Women," by Chas. B. Penrose, M.D., Ph.D. W. B. Saunders, Philadelphia, 1897.

"The Diseases of Women." A Handbook for Students and Practitioners, by J. Bland Sutton, F.R.C.S., and Arthur E. Giles, M.D., F.R.C.S., Edinburgh. W. B. Saunders, Philadelphia, 1897.

"The Menopause," by Andrew F. Currier, A.B., M.D. D. Appleton & Co., New York, 1897.

"The Eye as an Aid in General Diagnosis," by E. H. Linnell, M.D., The Edwards & Decker Co., Philadelphia, 1897.

"A New Classification of the Motor Anomalies of the Eye," by Alexander Duane, M.D. J. H. Vail & Co., New York, 1897.

"Tuberculosis of the Genito-Urinary Organs of Male and Female," by N. Senn, M.D., Ph.D., LL.D. W. B. Saunders, Philadelphia, 1897.

"Hypnotism and its Application to Practical Medicine," by Otto Geo. Wetterstrand, M.D., translated by Henrik G. Petersen, M.D. Putnam's Sons, New York, 1897.

"Normal and Pathological Circulation of the Central Nervous System," by Wm. Browning, Ph.B., M.D. The Lippincott Co., Philadelphia, 1897.

"Traumatic Injuries of the Brain and its Membranes," by Chas. Phelps, M.D. D. Appleton & Co., New York, 1897.

"The Psychology of the Emotions," by T. Ribot. Walter Scott, London, Eng., 1897.

"Crime and Criminals," by J. Sanderson Christison, M.D. The W. T. Kenner Co., Chicago, 1897.

"The Insane in Private Dwellings and Licensed Houses," by J. F. Sutherland, M.D., F.R.S.E., 2d edition. E. and S. Livingstone, Edinburgh, Eng.

"Proceedings of the Fifty-second Annual Meeting of the American Medico-Psychological Association, held in Boston, May 26-29, 1896. By the American Psychological Association, 1896.

"Vol. II. of 'A System of Practical Medicine' by American Authors." Edited by Alfred L. Loomis, M.D., LL.D., and Wm. Gilman Thompson, M.D. Lea Bros. & Co., N. Y., 1897.

"Syringomyelia," by Guy Hinsdale, A.M., M.D. The International Medical Magazine Co., 1897.

"Annals of Otology, Rhinology and Laryngology" (quarterly). Published by J. H. Parker, St. Louis, Mo., August, 1897.

"Pathological Technique," a Manual for Pathological Laboratory, by Drs. F. B. Mallory and J. H. Wright. W. B. Saunders, Philadelphia, 1897.

"A Text Book of the Practice of Medicine," by Jas. M. Anders, M.D. W. B. Saunders, Philadelphia, 1897.

"Essentials of Bacteriology," by M. V. Ball, M.D. W. B. Saunders, Philadelphia, 1897.

"Arbeiten aus dem Gesamtgebiet der Psychiatrie und Neuro-pathologie." von R. v. Krafft-Ebing. II. Heft. Johann Ambrosius Barth, Leipzig, 1897.

"A Text Book of Mental Diseases," by Theo. H. Kellogg, A.M., M.D. Wm. Wood & Co., New York, 1897.

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This standard prepared food for invalids and children has won the enviable distinction of having successfully stood the crucial test of years of actual clinical experience in private practice, sanitariums and hospitals, while numerous competing preparations have appeared and disappeared—often so completely that even their names are forgotten. The Imperial Granum, however, enjoys so universally the confidence of physicians that its merits are beyond dispute. Moreover, the decisions of its manufacturers not to publicly advertise it has secured for it the endorsement of even the most ethical members of the medical profession, who dislike to prescribe any article advertised broadcast to the people and profession alike. Physicians can obtain sample packages free, charges prepaid, on application to the Imperial Granum Co., New Haven, Ct., or John Carle & Sons, New York City.

OPHTHALMIA NEONATORUM.

Dr. E. F. West, of San Francisco, Cal., writes, "I have used Palpebrine with excellent results in Ophthalmia Neonatorum of severe types. Cleansing the eyes with a 25 per cent. solution every half hour. A few drops of full strength applied four times a day."

SOMETHING NEW.

In an article on "Formaldehyde Disinfection," published by Dr. S. Rideal, Lecturer at St. George's Hospital, London, in the November issue of Public Health, the journal of the incorporated society of medical officers of health of England, the author concludes as follows: "It will be seen from the above experiments that I have obtained sufficient good results with 10 grammes (10 pastils) per 1,000 cubic feet to warrant this quantity being used in all cases of ordinary disinfection, and if in special cases the walls and floors are in addition sprayed with a 0.5 per cent. Formalin solution before using the lamp, I believe that the best practical means of disinfection would be in this way assured."

QUITE IN LINE.

The substitution of one article for another is a crime alike against physician and patient. The medical profession can put an end to it by sending their prescriptions only to those pharmacists around whom there rests not the slightest suspicion.

SOLUBILITY.

"If it's a pill made by William R. Warner & Co., it's soluble." There's only a few words in the above sentence, yet they are words which will impress all who read them. The theme is not a new one. The name "Warner" has long had the word "solubility" intimately associated with it. While we congratulate Messrs. Wm. R. Warner & Co. upon the perfect preparations bearing their name, we cannot but state we do not see why they should not be perfect. Forty-one years in business constitutes a period, during which a progressive house should be able to give to the profession perfect preparations. The pills made thirty years ago and exhibited at the American Medical Convention, in Philadelphia, proved to be as perfect and soluble as the day they were made. The following is suggestive that Messrs. Wm. R. Warner & Co. have been very successful:

Liberty Ohio, June 9, 1897.

Messrs. WM. R. WARNER & Co., Philadelphia:

Gentlemen—Last winter I unearthed a small vial of your Aloin Granules that by chance had been stowed away for *twelve years*. Having always used your Aloin Granules in my practice, I, of course, used these, and, as far as I could determine, *they were as efficient as the day they were made*. I tried them on myself several times with results as good as could be wished for. I have kept a few as a curiosity. They are O. K. Yours truly,

J. H. ADAIR.

SANMETTO IN BRIGHT'S DISEASE.

Charles F. Reiff, M.D., of Fremont, O., writing, says: "I prescribed Sanmetto in a case of advanced Bright's Disease. The patient became more comfortable, and since then has used several bottles of Sanmetto. In my opinion Sanmetto is the most efficient remedy for diseases of the genito-urinary organs, and I shall continue to prescribe the remedy."

WORTH NOTICE.

If the druggist found that every attempt at substitution cost him the physician's patronage, he would soon become tired of it, and would supply exactly what prescriptions call for.

AN ANTIDOTE TO THE TWO GREAT SYMPTOMS.

The value of Antikamnia consists in its rapid effect in alleviating the suffering of the patient while more radical treatment is working a cure. While endeavoring to rid our patient of his neuralgia, rheumatism, typhoid, intermittent or malarial fever, we secure him relief from pain and intermission of fever. We have, in short, in this drug, not a remedy for any disease, but a most useful antidote to the two great symptoms—pain and pyrexia.—Medical Reprints, London, Eng.

MALTZYME WITH HYPOPHOSPHITES.

Formula: each fluid ounce contains—Calcium Hypophosphite, 4 grs.; Potassium Hypophosphite, 4 grs.; Ferric Hypophosphite, $\frac{1}{4}$ gr.; Manganese Hypophosphite, $\frac{1}{4}$ gr.

The profession has long recognized the great value of the hypophosphites in certain conditions, and in recent years the addition of manganese has been universally accepted as advisable. Unlike other hypophosphite preparations, the base of "Maltzyme with Hypophosphites" is not an inert syrup, but Maltzyme, which promotes the assimilation of starchy foods. Hence, the patient secures not only the therapeutic action of the hypophosphites, but also of the food constituents and distaste of Maltzyme.

JACQUEMAIRE'S GLYCEROPHOSPHATES *Vital Phosphate*

RECONSTRUCTIVE AGENT OF THE NERVOUS SYSTEM. THE ONLY ONE EXPERIMENTED WITH AT THE PARIS HOSPITALS.

N. B.—They are the BASIS of Dr. Albert Robin's Communication to the French Académie de Médecine, on April 24th, 1896.

FORMS:
1st. Vital Phosphate of Lime, Granulated
2d. Vital Phosphate Comp. Granulated
a combination of the different phosphates of the bones
quartz, lime, magnesia, potash, soda and iron.
Strength: 4½ grains to the teaspoon.

INDICATION:
Children: adolescence;
Diseases of the bones.
Nervous exhaustion, neurasthenia,
phosphoric albuminuria, chlorosis,
acute catarrhs, rheumatism, etc.

Price per bottle, { Granulated, of Lime, . . . \$1.00
Granulated, Comp. . . . \$1.25

Phosphoglycerates being precipitated by alcohol, their use in medicinal wines is not advisable. The best form for administration is the granulated.

LITERATURE ON APPLICATION. THE CARABAÑA CO., 2-4 Stone St., New York, Sole Agents for the United States.

At the head

of all of the best
Aperient Waters
stands 

Carabaña

At all Druggists.

It is superior to all Hunyadi's. Takes a smaller dose to produce better results, while at the same time it tastes better.

Sole Importers, THE CARABAÑA COMPANY, 2 Stone Street, New York.

SANMETTO FOR GENITO-URINARY DISEASES.

A Scientific Blending of True Santal and Saw Palmetto In a Pleasant Aromatic Vehicle.

A Vitalizing Tonic to the Reproductive System.

**SPECIALLY VALUABLE IN
PROSTATIC TROUBLES OF OLD MEN—IRRITABLE BLADDER—
CYSTITIS—URETHRITIS—PRE-SENILITY.**

DOSE:—One Teaspoonful Four Times a Day.

OD CHEM. CO., NEW YORK.

THE UNIVERSAL

MULTI-NEBULAR VAPORIZER FOR OFFICE USE

In the treatment of all diseases of the

Respiratory Organs and Middle Ear

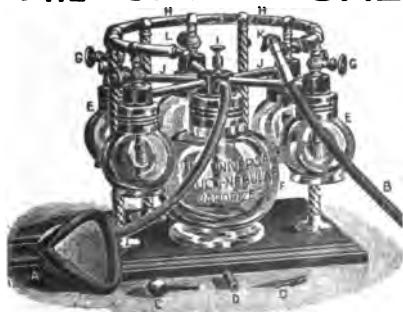
by Ten Different Methods, including

**VAPO-PULMONARY MASSAGE
and VAPO-AURAL MASSAGE**

Is Indispensable in Office Practice

Write for circular describing the instrument
and methods of use.

GLOBE MFG. CO.
Battle Creek, Mich.



XXIV.

ALWAYS RELIABLE.

NO DETRIMENTAL AFTER-EFFECTS.

It has been proven by clinical tests that *Neurostine* is the most effective and safest hypnotic yet known to the profession, whereas it contains no morphine, chloral or opium, there can be no detrimental after-effects. Always of the same consistency, therefore may be relied upon to produce the same results under similar conditions. It is only necessary for physicians to give *Neurostine* a trial and they will be convinced that it is *the standard remedy* in the treatment of all forms of nervous disturbances. In uterine troubles it should be combined with Dioiburnia.

Dose: One teaspoon to a tablespoonful three or more times a day, as indicated.

DIOS CHEMICAL CO., ST. LOUIS.

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You Get the Profits

Of Dealers, Agents, Jobbers
and Middlemen by buying direct
from the manufacturer.



No better wheel made than the

Acme Bicycle

Built in our own factory by
skilled workmen, using the best
material and the most improved
machinery. *We have no agents*
Sold direct from factory to the
rider, fully warranted. Shipped
anywhere for examination.

WRITE FOR

Our Interesting Offer

Acme Cycle Co., Elkhart, Ind.

ARE YOU USING ~ ~ ~ ~

Peptenzyme?

Peptenzyme

Is the only perfect digestant.

Digests every kind of food, albumen, fat, starch, cane sugar, reducing them to the exact conditions required for assimilation in the organism.

Presents in physiological activity the digestive principles, active and embryo ferments, from all the digestive glands.

Is the only preparation which contains the enzymes isolated by a mechanical process, and unchanged from the condition as found in the living gland.

Peptenzyme is far superior to any other preparation in the treatment of all disorders of the digestive organs. It promotes digestion, both by aiding and perfecting the process itself and by stimulating the appetite and secretory functions through the absorption of the embryo ferments. It not only gives immediate relief, but aids in *curing* Dyspepsia, etc. Pepsin, as found in the market, is prepared only by chemical methods, and has consequently lost most of its physiological properties, and is of little service in aiding digestion.

Peptenzyme is prepared in three forms, Elixir, Powder and Tablets.

SAMPLES, LITERATURE AND DIET LEAFLETS UPON REQUEST.

REED & CARRICK, ~ NEW YORK

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YOU
WANT

The Erie Bicycle

It has the best seamless tubing.

It has detachable sprockets, both front and rear.

Its fork crown has never been known to bend or break.

It has all the modern equipment, including best tires.

Its cranks are square, exceedingly strong and reliable.

Its price is within the reach of all, \$75.

You can get no better wheel at any price.

Send for Catalogue.

QUEEN CITY CYCLE CO., Idlewood, N.Y.

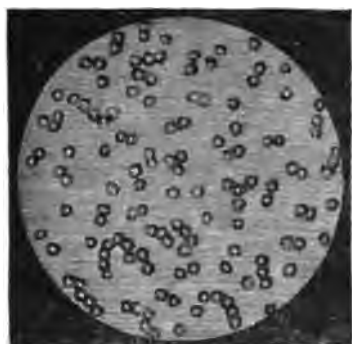
THE CROWNING DEVELOPMENT OF PRACTICAL MEDICINE

IN HÆMATHERAPY, OR BLOOD TREATMENT.

BLOOD, AND BLOOD ALONE, is physiologically ascertained to be the essential and fundamental Principle of Healing, of Defense, and of Repair, in the human system; and this Principle is now proved, by constant clinical experience, to be practically available to the system in all cases, to any extent, and wherever needed, internally or externally.

And the same overwhelming clinical demonstrations have also proved that the Vitality and Power of Bovine Blood can be and are *PRESERVED*, unimpaired, in a portable and durable preparation, sold by all druggists, and known as Bovinine. Microscopic examination of a film of Bovinine will show the **LIVING BLOOD CORPUSCLES** filling the field, in all their integrity, fullness, and energy; ready for direct transfusion into the system by any and every mode of access known to medical and surgical practice; alimentary, rectal, hypodermical, or topical.

A FILM OF BOVININE:
Showing the Blood-corpuscles Intact.



Micro-photographed
by Prof. R. R. Andrews, M.D.

In short, it is now an established fact, that if Nature fails to *make* good blood, *we* can *introduce* it. Nothing of disease, so far, has seemed to stand before it.

Apart from private considerations, these facts are too momentous to mankind, and now too well established, to allow any further reserve or hesitation in asserting them to the fullest extent.

We have already duly waited, for three years; allowing professional experimentation to go on, far and near, through the disinterested enthusiasm which the subject had awakened in a number of able physicians and surgeons, and these daily reinforced by others, through correspondence, and by comparison and accumulation of their experiences in a single medical medium adopted for that provisional purpose.

It is now laid upon the conscience of every physician, surgeon, and medical instructor, to ascertain for himself whether these things are so; and if so, to develop, practise and propagate the great medical evangel, without reserve. They may use our Bovinine for their investigations, if they cannot do better, and we will cheerfully afford every assistance, through samples, together with a profusion of authentic clinical precedents, given in detail, for their instruction in the philosophy, methods and technique of the New Treatment of all kinds of disease by Bovine Blood, so far as now or hereafter developed.

☛ Among the formidable diseases overcome by the Blood Treatment, in cases hitherto desperate of cure, may be mentioned: Advanced Consumption; Typhoid Fever; Pernicious Anæmia; Cholera Infantum, Infantum, etc.; Hæmorrhagic Collapse; Ulcers of many years standing, all kinds; Abscesses; Fistulas; Gangrene; Gonorrhœa, etc.; Blood-poisoning; Crushed or Decayed Bones; Mangled Flesh, and great Burns, with Skin-propagation from 'points' of skin; etc., etc.

N. B. Bovinine is not intended to be, and cannot be made, an article of popular self-prescription. As it is not a stimulant, its extended employment in the past has been, and the universal employment to which it is destined will be, dependent altogether on the express authority of attending physicians. Address

THE BOVININE COMPANY, 495 WEST BROADWAY, NEW YORK.

Maltine

MALTINE is not merely "malt," nor is it a mere "extract of malt," nor an "essence of malt."

MALTINE is the most highly concentrated extraction of all the nutritive and digestive properties of Wheat, Oats and Malted Barley.

It has stood alone and unrivalled throughout the world in its therapeutic field for more than twenty years, despite the most strenuous efforts of the ablest pharmaceutical manufacturers to produce a preparation approaching it in medicinal value, elegance, palatability and stability.

"Malt" is *not* "MALTINE."

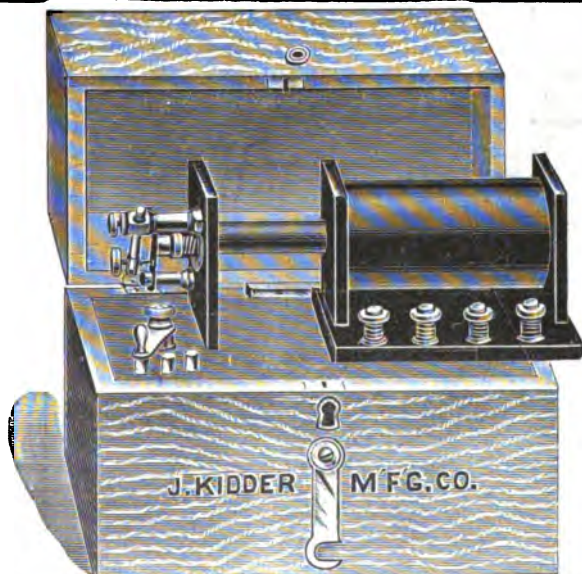
"Extract of Malt" is *not* "MALTINE."

"Essence of Malt" is *not* "MALTINE."

"**MALTINE**" must be designated to get
"MALTINE."

A NEW FARADIC APPARATUS.

COIL CONTAINS 4,500 FT. OF WIRE.



820 BROADWAY, NEW YORK, N. Y.

JEROME KIDDER M.F.G. CO.

SPECIAL APPARATUS No. 7.

SINGLE SPOOL, 1,500 YARDS—OPERATED BY DRY CELLS.

In polished walnut case $8\frac{3}{4}$ inches long, $6\frac{1}{4}$ inches wide, $7\frac{1}{4}$ inches deep, outside measurements. Containing spool of No. 32 wire, 1,500 yards (4,500 ft.) long, wound in sections of 300, 500 and 700 yards, producing six different combinations. Current graduated from absolutely nothing to the desired strength. The induced coil is arranged to be removed entirely from the primary field, thus grading the current from absolutely nothing to the desired strength of each combination. The apparatus is most compact and covers a field of usefulness where a medium high tension current is desired at a moderate cost.

DR. McMUNN'S ELIXIR OF OPIUM.

An Invaluable Discovery in the preparation of Opium.

It contains all the valuable medicinal properties of Opium in natural combination, to the exclusion of all its noxious, deleterious, useless principles upon which its bad effects depend. It possesses all the sedative, anodyne and antispasmodic powers of Opium: To produce sleep and composure; to relieve pain and irritation, nervous excitement and morbid irritability of body and mind; to allay convulsive and spasmodic actions, etc.; and being purified from all the noxious and deleterious elements, its operation is attended by no sickness of the stomach, no vomiting, no costiveness, no headache, nor any derangement of the constitution or general health. Hence its superiority over Laudanum, Paregoric, Black Drop, Denarcotized Laudanum, and every other Opiate preparation.

CAUTION.

On account of its large sale, spurious articles are offered in bulk. The genuine is sold only in vials of about 7 drachms, with yellow wrappers and signature of Jno. B. McMunn.

E. FERRETT, Agent, 372 Pearl St., New York.

PRESCRIBE DR BRUSH'S KUMYSS

WHY
EXPERIMENT
WITH
IMITATIONS?

A SCIENTIFIC PRODUCT PROFESSIONALLY GUARDED
FROM START TO FINISH.

A Remedy in Nervous Disorders when
Characterized by Melancholia.

—Mode of Exhibition.—

The "Reference Book of Practical Therapeutics," by Frank P. Foster, M. D., Editor of *The New York Medical Journal*, which has recently been issued by D. Appleton Co., of New York City, contains an article of which the following is an excerpt, which we feel expresses the consensus of medical opinion as adduced by actual results: "Antikamnia is an American preparation that has come into extensive use as an analgetic and antipyretic. It is a white, crystalline, odorless powder, having a slightly aromatic taste, soluble in hot water, almost insoluble in cold water, but more fully soluble in alcohol.

"As an antipyretic it acts rather more slowly than antipyrine or acetanilide, but efficiently, and it has the advantage of being free, or almost free from any depressing effect on the heart. Some observers even think that it exerts a sustaining action on the circulation. As an analgetic it is characterized by promptness of action and freedom from the disagreeable effects of the

narcotics. It has been much used, and with very favorable results in neuralgia, influenza and various nervous disorders characterized by melancholia. The dose of antikamnia is from three to ten grains, and it is most conveniently given in the form of tablets."

We may add, that the best vehicles, in our experience, for the exhibition of antikamnia are Simple Elixir, Adjuvant Elixir or Aromatic Elixir, as also brandy, wine or whiskey. It can also be readily given in cachets or capsules, but preferably tablets, as well as dry on the tongue in powder form, followed by a swallow of water. When dispensed in cachets or capsules it should be put into them dry. Antikamnia tablets should be crushed when very prompt effect is desired and patients should always be so instructed. The conditions of the stomach frequently present unfavorable solvent influences and they can be thus overcome.

—Notes New Pharm. Products.

In Pneumonia where there is Restlessness.

R Antikamnia (Genuine).....	3 ij
Tinct. Digitalis.....	3 iss
Syrup Doveri.....	3 iij
Mx. Sig. :—Teaspoonful every 3 to 6 hours.	

In Painful Dysmenorrhœa.

R Antikamnia (Genuine).....	3 j
Brom. Potass.....	3 ij
Elix. Aurantii.....	3 ij

Mx. Sig. :—One or two teaspoonfuls every hour in water. —Dunghison's Clinical Record.

VALUABLE SPECIFIC.

C. A. Bryce, A.M., M.D., Professor of Surgery in the National College, Indianapolis, Ind.; Surgeon-in-charge "The Hickories," Editor Southern Clinic, Author "Bryce's Practice of Medicine," etc., etc., writes: "There came to my office in March, 1897, a man, about 28 years of age, with the most extensive multiple ulcers of the legs that I ever saw. He said that he had tried for months to get relief without benefit, and desired me to do what I could for him. His legs, from the knees to the ankles, were swollen and infiltrated. The entire surface was undergoing destructive changes, and circle-corroding ulcers were in all stages—all of them finally cutting cleanly through the skin down to the muscle. The case was clearly one of the late manifestations of syphilis implanted upon a scrofulous base. Locally, I used a cleansing and gently stimulating, protective treatment, and placed the patient upon Elixir Iodo of Bromide of Calcium Compound (Tilden), which he continued to use without intermission for over two months, with the result of effecting a complete cure. I gave him positively nothing at any time but the Elixir Iodo, and attribute his cure entirely to its great alterative properties and special adaptability to cases of this character. The results were simply astounding. I have used Elixir Iodo Bromide of Calcium Compound (Tilden) in a case of syphilitic lung involvement, curing the patient after he had been given up to die by an excellent practitioner."

OPINIONS ON SCHERING'S FORMALIN-DISINFECTION METHOD.

1. Professor C. Flügge, of Breslau, Geheimer Medizinalrath and Member of the Imperial Health Department, writes: "I regard Formalin disinfection as very valuable in various infectious diseases. As can be seen from the accompanying abstract of a communication from Dr. E. Poleck, I believe that your apparatus, after comparison with those of Trillat and of Rosenberg, is the most practically useful one. It is my intention to employ it in the near future upon a larger scale, more especially since, by order of the "Cultusminister" a School of Disinfection is to be established here."

2. Dr. Alexander, Regierungs- and Medizinalrath, of Breslau, has written to inform us that the value of our Formalin Disinfection method is well recognized in the Breslau Hygienic Institute.

3. Dr. E. Polleck, of Breslau, Assistant in the Hygienic Institute, at the conclusion of a paper read before the Section on Hygiene of the "Schlesische Gesellschaft für vaterländische Cultur," July, 28, 1897, says as follows: (Allgemeine medizinische Centralzeitung, No. 66, 1897): "Aronson's results have been tested by Dr. Laschtschenkow in the Hygienic Institute here, and have been fully confirmed."

The Schering apparatus is cheap and handy, requires no attention whilst in use, and the results are good and reliable; it is, therefore, positively to be preferred to that of Trillat.

4. Dr. Markl, K.K. Bezirksarzt in the Ministry of the Interior, Vienna, writes: "All the experiments made with Schering's Formalin Lamp, which you have sent us, have been successful."

HINT THAT OUGHT TO TELL.

The medical profession recognize the necessity of correct diagnosis, but should not forget that the public estimate the doctor's skill, not by his familiarity with technical details, but by actual results. See to it that your prescriptions are filled as written.

XXX,

SYR. HYPOPHOS. CO., FELLOWS

Contains the Essential Elements of the Animal Organization—Potash and Lime;

The Oxydizing Agents—Iron and Manganese;

The Tonics—Quinine and Strychnine;

And the Vitalizing Constituent—Phosphorus; the whole combined in the form of a Syrup with a Slightly Alkaline Reaction.

It Differs in its Effects from all Analogous Preparations; and it possesses the important properties of being pleasant to the taste, easily borne by the stomach, and harmless under prolonged use.

It has Gained a Wide Reputation, particularly in the treatment of Pulmonary Tuberculosis, Chronic Bronchitis, and other affections of the respiratory organs. It has also been employed with much success in various nervous and debilitating diseases.

Its Curative Power is largely attributable to its stimulant, tonic, and nutritive properties, by means of which the energy of the system is recruited.

Its Action is Prompt; it stimulates the appetite and the digestion, it promotes assimilation, and it enters directly into the circulation with the food products.

The prescribed dose produces a feeling of buoyancy, and removes depression and melancholy; hence the preparation is of great value in the treatment of mental and nervous affections. From the fact, also, that it exerts a double tonic influence, and induces a healthy flow of the secretions, its use is indicated in a wide range of diseases.

NOTICE—CAUTION.

The success of Fellows' Syrup of Hypophosphites has tempted certain persons to offer imitations of it for sale. Mr. Fellows, who has examined samples of several of these, finds that not two of them are identical, and that all of them differ from the original in composition, in freedom from acid reaction, in susceptibility to the effects of oxygen when exposed to light or heat, in the property of retaining the strychnine in solution, and in the medicinal effects.

As these cheap and inefficient substitutes are frequently dispensed instead of the genuine preparation, physicians are earnestly requested, when prescribing the Syrup, to write "Syr. Hypophos. Fellows."

As a further precaution, it is advisable that the Syrup should be ordered in the original bottles; the distinguishing marks which the bottles (and the wrappers surrounding them) bear, can then be examined, and the genuineness—or otherwise—of the contents thereby proved.

Medical Letters may be addressed to:

Mr. FELLOWS, 48 Vesey St., New York.

Of Importance to Physicians:

We beg to call the attention of Physicians to the fact that the makers of

Phenacetine-Bayer, Sulfonal-Bayer, and Trional

are supplying these products in

Dosed Powders of 10 grains each

as well as in ounce cartons, as formerly. These powders are put up in ounce cartons containing 44 ten grain powders, each powder having the seal of the makers on the cutting line. In many instances it will be found advantageous to prescribe these remedies in these original powders, thereby insuring the genuine character of the products. When you desire these powders dispensed be sure and specify them in your prescriptions.

Full descriptive pamphlets mailed by

**Schieffelin & Co., New York,
Sole Agents for Pharmaceutical Products of Farbenfabriken
vorm. Friedr. Bayer & Co., Elberfeld, Germany.**

**In the treatment of
diarrhoeal affections,
whether acute or chronic,**

TANNIGEN

has proved of
exceptional value.

Tannigen, or acetyl tannin, is an ideal intestinal astringent, which passes unchanged through the stomach and is decomposed gradually in the intestine, exerting its specific action upon the entire intestinal canal. It is odorless, tasteless, agreeable of administration, and perfectly innocuous and unirritating. In acute and chronic diarrhoeal troubles, both of children and adults, dysentery, the diarrhoea of phthisis, Tannigen has been successfully employed.

ALSO,

**Piperazine-Bayer, Somatose, Sulfonal-Bayer, Phenacetine-Bayer,
Lycetol, Salophen, Iodothyrene, Aristol, Trional, and Losophan.**

Write for pamphlets to

**Schieffelin & Co., New York,
Sole Agents for Products of
Farbenfabriken vorm. Friedr. Bayer & Co., Elberfeld, Germany.**

Wm. R. Warner & Company's

SOLUBLE AND RELIABLE PHOSPHOROUS PILLS.



Pil: Phosphori, 1-100 gr. or 1-25 gr. (W. R. Warner & Co.)

DOSE—One pill, two or three times a day, at meals.

THERAPEUTICS—When deemed expedient to prescribe phosphorus alone, these pills will constitute a convenient and safe method of administering it.

Pil: Phosphori, Comp. (W. R. Warner & Co.)

R Phosphori, 1-100 gr.; Ext. Nucis Vomicae, $\frac{1}{4}$ gr.

DOSE—One or two pills, to be taken three times a day, after meals.

THERAPEUTICS—As a nerve tonic and stimulant this form of pill is well adapted for such nervous disorders as are associated with impaired nutrition and spinal debility, increasing the appetite and stimulating the digestion.

Pil: Phosphori cum Ferri et Nuc. Vom. (W. R. Warner & Co.)

R Phosphori, 1-100 gr.; Ferri Carb., 1 gr.; Ext. Nucis Vomicae, $\frac{1}{4}$ gr.

DOSE—One or two pills, may be taken two or three times a day, at meals.

THERAPEUTICS—This pill is applicable to conditions referred to in the previous paragraphs as well as to anemic conditions generally, to sexual weakness, neuralgia, in dissipated patients, etc.

Pil: Phosphori cum Ferro et Quinia et Nuc. Vom. (W. R. Warner & Co.)

R Phosph., 1-100 gr.; Ferri Carb., 1 gr.; Ext. Nuc. Vom., $\frac{1}{4}$ gr.; Qui. Sulph., 1 gr.

DOSE—One pill, to be taken three times a day, at meals.

THERAPEUTICS—The therapeutic action of this combination of tonics, augmented by the specific effect of Phosphorous, on the nervous system, may readily be appreciated.

Pil: Phosphori cum Cannabe Indica. (W. R. Warner & Co.)

R Phosphori, 1-50 gr.; Ext. Cannabis Indica, $\frac{1}{4}$ gr.

DOSE—One or two pills, to be taken twice or three times a day, at meals.

THERAPEUTICS—The Indian Hemp is added as a calmative and soporific in cases in which morphia is inadmissible from idiosyncrasy or other cause, as well as for its aphrodisiac effects.

WILLIAM R. WARNER & CO.,

Manufacturers of Reliable, Soluble-Coated Pills, etc.
PHILADELPHIA. NEW YORK.

Bone Marrow

As a Blood Maker.

The kind of marrow that makes **Red Corpuscles** is taken from the rib ends of calves and is hard to obtain in quantity, even in an abattoir as large as ours. For this reason we believe physicians will get better results in treating Anæmia and all diseases that are due to a depraved condition of the blood from the administration of Armour's **Extract of Red Bone Marrow** than from preparations manufactured by firms who buy supplies where and whenever they can.

The Armour Laboratory Products are all made from absolutely fresh material.

Samples to physicians upon request.

Armour & Company, Chicago.



